AUG 5 1927

VOLUME 15 NUMBER 2

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AUGUST, 1927

PUBLISHED MONTHLY BY AMERICAN MEDICAL ASSOCIATION, 535 NORTH DEARBORN STREET, CHICAGO, ILLINOIS. ANNUAL SUBSCRIPTION, $8.00

Entered as Second-Class Matter, July 10, 1925, at the Postoffice at Chicago, Illinois, Under the Act of March 3, 1879. Acceptance for mailing at special rate of postage provided for in Section 1103, Act of Oct. 3, 1917, authorized July 29, 1920.

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ARCHIVES OF SURGERY

VOLUME 15 AUGUST, 1927 NUMBER 2

POSTOPERATIVE PULMONARY ATELECTASIS *

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Our purpose in this presentation is to emphasize the fact that pulmonary atelectasis, partial or massive, is a common postoperative pulmonary complication and presents a striking and characteristic roentgenologic and clinical picture. No doubt the majority of those postoperative conditions which have been regarded previously as either aspiration or postoperative pneumonia, bronchitis or bronchopneumonia, have been cases of atelectasis. In other words, the rarity of this condition, as judged by the number of reported cases, is apparent and not real.

DEFINITION

Postoperative pulmonary atelectasis is a condition in which deflation occurs in a previously well-aerated lung. This condition comes on in from twelve to forty-eight hours after operation and involves the entire lung or any part of it.

Jackson and Lee [1] suggest the term "atelectasis" as being more accurately descriptive of this condition, and hold that the term "collapse" should be reserved for those cases caused by an increased intrapleural pressure. In atelectasis, the bronchi are usually not involved; in collapse, the bronchi are collapsed along with the alveolar tissues.

HISTORICAL RESUMÉ

In 1890, William Pasteur [2] of England first described massive collapse occurring in postdiphtheritic cases, with paralysis of the diaphragm in thirty-four instances. With the knowledge thus acquired, he [3] was able to recognize the postoperative variety and described sixteen cases occur-

* From the Surgical and Roentgenological Departments, St. Alexis Hospital.

1. Jackson, Chevalier, and Lee, W. E.: Ann. Surg. **82**:364 (Sept.) 1925.
2. Pasteur, William: Internat. J. M. Sc., Sept. 1890, p. 242.
3. Pasteur, William: Brit. J. Surg. **1**:587, 1914.

ring in 2,000 abdominal operations in 1914. In the same year, Elliott and Dingley [4] reported eleven cases following abdominal operations.

During the period between 1918 and 1920, Bradford [5] studied massive collapse in connection with war wounds.

Scrimger [6] was the first to describe this condition in the American literature. In 1921, he reported seven postoperative cases, four of which followed appendectomy; two, herinorrhaphy, and one, hemorrhoidectomy.

In 1925, Scott [7] reviewed all the reported cases of the postoperative variety and found sixty-four cases, to which he added four of his own. In September of the same year, Jackson and Lee [1] demonstrated the relationship between massive atelectasis and plugging of the bronchi by foreign bodies or secretion, the removal of which leads to a gradual reinflation of the collapsed lung.

THEORY

Four theories have been evolved as a result of the observations and investigations conducted by various authors:

1. In 1908, Pasteur [8] advanced the opinion that collapse of the wall of the chest, due to paralysis of the diaphragm and, probably, of the accessory respiratory muscles, was primary and that the collapse of the lung was secondary. Subsequent investigators, adhering to this original opinion, added the statement that any bronchial secretion which may be found is present merely as a result of diminished aeration and may aid the process. Bradford [5] was a supporter of this idea. The observations of Sante [9] do not support this theory.

2. Elliott and Dingley [4] failed to obtain atelectasis in cats, when they produced hemiparalysis of the diaphragm and the respiratory musculature. They advanced the theory that atelectasis was produced as a result of diaphragmatic immobilization associated with bronchial obstruction, the alveolar air being absorbed by the circulating blood. Jackson and Lee [1] and Tucker [10] favor this explanation and maintain that obstruction alone, produced by inspissated mucus plugs, may cause atelectasis.

3. Brisco [11] considered the atelectasis secondary to inflammation affecting the retroperitoneal portion of the diaphragm. This results in

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4. Elliott, T. R., and Dingley, L. A.: Lancet **1**:1305, 1914.
5. Bradford, Sir J. R.: Oxford Loose-Leaf Medicine **2**:127, 1920.
6. Scrimger, F. A. C.: Surg. Gynec. Obst. **32**:486 (June) 1921.
7. Scott, W. J. M.: Postoperative Massive Collapse of Lung, Arch. Surg. **10**:73 (Jan.) 1925.
8. Pasteur, William: Bradshaw Lecture, Royal College of Physicians, 1908.
9. Sante, L. R.: Radiology **8**:1 (Jan.) 1927.
10. Tucker, Gabriel: Surg. Gynec. Obst. **42**:743, 1926.
11. Briscoe, J. C.: Quart. J. Med. **8**:293, 1920.

a disturbance of the function of the diaphragm and the associated respiratory muscles on the involved side.

4. Bradford,[5] although favoring the theory of Pasteur,[8] suggested that the condition could be brought about by vagal stimulation affecting the constrictor mechanism of the bronchioles, but he could not offer any corroborative evidence. Scott[7] favors this theory and considers that atelectasis is produced by means of a vasomotor mechanism, the atelectasis being primary and the bronchial obstruction secondary. A thick, tenacious mucus present in the bronchi, due to the stimulation of a low grade infection, is the cause of the obstruction. Sante[9] added that the force of cohesion held the walls of the bronchioles in apposition, once they were approximated. Trout and Hayter[12] are of the opinion that anesthetics do not play a part in the production of this complication and also give support to the theory of Bradford.[5]

The contralateral variety of atelectasis following slight trauma or fracture, as noted by Bradford,[5] is best explained by the fourth theory. The experimental work of Carlson[13] has been cited as supportive of this theory. This author produced contraction and expansion of the lungs of certain amphibians by stimulation of the vagus and demonstrated the same effect by electrical and mechanical stimulation of peripheral nerves in various parts of the body.

In general, we agree with Elliott and Dingley;[4] but, from our observations, we believe that partial atelectasis is more common than the massive type and that the causative factors are of gradual development, being present for some time before the condition manifests itself.

The following factors are operative in the production of atelectasis:

1. Diaphragmatic fatigue.

2. Diaphragmatic inhibition:

(*a*) The recumbent posture, which nearly all postoperative patients are made to assume, limits the facility of respiration and reduces the vital capacity (MacLeod).[14]

(*b*) Splinting of the diaphragm and the accessory respiratory muscles on the affected side as a protective reflex occasioned by recent operative intervention tends further to decrease aeration or tidal air.

3. Bronchial obstruction.

(*a*) Decreased aeration produces diminution in evaporation of bronchial secretion, with gradual increase in bulk. It has been noted by various observers and by ourselves that many patients presenting

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12. Trout, H. H., and Hayter, H. M.: Virginia M. Monthly **53**:214, 1926.

13. Carlson, in Scrimger (footnote 6).

14. MacLeod, J. J. R.: Physiology and Biochemistry in Modern Medicine, St. Louis, C. V. Mosby Co., 1920, p. 316.

a mild bronchitis prior to operation develop an extraordinary degree of bronchorrhea postoperatively, even after spinal, local or gas anesthesia.

(*b*) The cough reflex is in abeyance, due partly to the use of morphine and partly to the voluntary postoperative inhibition of muscular activity permitting accumulation of secretion.

(*c*) When the obstruction is complete in any bronchus, the imprisoned air is absorbed by the circulating blood, and atelectasis ensues.

In further support of our belief, the following facts are offered:

1. Physical examination of patients immediately following operation and at various intervals thereafter has shown that atelectasis does not occur until several hours have elapsed. This argues against the sudden production of atelectasis by means of a reflex vagus mechanism and shows that time must elapse during which there is an accumulation of mucus leading to bronchial obstruction.

2. In a number of instances, we have been able to anticipate the development of atelectasis in patients under anesthesia, who gave evidence of increased secretion in the respiratory tract. We have applied the term "wet patients" to these cases.

3. The finding of mucus plugs by Jackson and Lee [1] in two cases at necropsy, and the demonstration of such plugs in vivo. Nearly all observers have noted the presence of mucopurulent expectoration.

4. Reinflation of the atelectatic lung following bronchoscopic aspiration of the mucus, as described by Tucker [10] and Manges.[15]

5. Recurrence of atelectasis following reaccumulation of mucus in the bronchial tree, in amounts sufficient to produce obstruction. The cycle of secretion-obstruction-atelectasis-aspiration-reinflation has been completed seven times in one case, reported by Hearn and Clerf.[16] It would be difficult to explain this phenomenon on the basis of stimulation of the vagus.

6. Atelectasis of mild degree may be produced in normal persons by placing them for a time on their right side. However, this differs from the postoperative form in that it disappears immediately on a deep inspiration, as shown by fluoroscopic observations reported by Webb, Forster and Gilbert.[17]

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15. Manges, W. F.: Am. J. Roentgenol. **11**:517 (June) 1924.

16. Hearn, W. P., and Clerf, L. H.: Ann. Surg. **85**:54 (Jan.) 1927.

17. Webb, G. B.; Forster, A. M., and Gilbert, G. B.: Postural Rest for Pulmonary Tuberculosis, J. A. M. A. **76**:846 (March) 1921.

INCIDENCE

Partial or massive pulmonary atelectasis is encountered following all kinds of abdominal operations and after all types of anesthesia. We have observed cases develop after spinal, local infiltration and regional block anesthesia, nitrous oxide and oxygen and also after ether anesthesia administered by either the open or the closed method, with and without gas induction.

Pasteur [2] has reported cases associated with diphtheritic paralysis of the diaphragm.

Bradford [5] has reported massive atelectasis after gun-shot wounds of the chest, abdomen, thigh and buttocks, and following fractures of the pelvis and femur, and also has mentioned two cases in which atelectasis occurred as a sequel to ordinary pneumonia without fibrosis of the lung or thickening of the pleura.

Other authors have noticed this condition following infectious diseases in persons of various ages. In our series, the youngest was a boy, aged 12, and the oldest, a man, aged 68.

Thirty of the fifty patients were males. The greater number of females operated on makes the actual incidence 3:1. In a three month period, in which there were 419 major cases in which operations had been done, thirty-two patients developed atelectasis. This comprised at least 70 per cent of all postoperative pulmonary complications occurring within this period.

SITE OF ELECTION

The part of the lung involved in either the partial or massive form, according to frequency is: (1) The posterior portion of the right lower lobe; (2) the right lower and middle lobes; (3) the left lower lobe; (4) the right upper lobe and (5) the left upper lobe.

The anatomic peculiarity of the lungs and bronchi is the reason offered for the more common occurrence of this condition on the right side.

ONSET

The onset is usually indicated by one or several of the following signs and symptoms: Marked facial erythema; sensation of tightness or actual pain in the chest; dyspnea or tachypnea; sudden elevation in temperature, pulse and respiration; cough, with or without expectoration; profuse diaphoresis; cyanosis; displacement of the heart toward the affected side, and asymmetry of the chest.

The physical signs and the severity of the symptoms depend on the amount of lung involved. These may appear suddenly or may develop gradually over a period of several hours. The average time of onset in our series was twenty-four hours.

CLINICAL FORMS

From a consideration of the clinical manifestations presented in our cases, we have been able to recognize four types: (1) The fulminant or frank type; (2) the moderate type; (3) the latent or mild type and (4) the transient or evanescent type.

1. *Fulminant Type.*—The fulminant type is the most readily recognized, presenting a unique symptom complex. It represents those cases referred to by several authors as the "collapse attack." The attack begins with a sudden sharp rise in temperature, pulse and respiration, associated with a sharp pain in the chest, dyspnea and cyanosis. The patient appears moribund; however, there is an absence of toxicity unless this complication is associated with some other acute infectious process. These patients have, in addition, a profuse diaphoresis, which necessitates frequent changing of gowns and bed clothes, coupled with pronounced facial erythema, which assumes a brick-red discoloration, as described by Soltau.[18] The alarming symptoms usually abate within thirty-six hours, and the further course is identical with that of atelectasis of moderate severity.

A characteristic posture, which is almost diagnostic, is assumed in this stage. The dorsal spine is bent and the head is inclined toward the affected side. The patient lies on the side involved.

Physical Examination.—The postoperative examination of patients is carried out under unfavorable conditions, because the patient cannot be moved about, nor can he be made to assume a position favorable for the elicitation of certain signs.

Inspection, which should be carried out with the patient as straight as possible in bed and lying flat on his back, reveals the marked asymmetry of the chest. The affected side appears smaller, as if flattened; the interspaces are diminished in size; the ribs seem to converge, and a marked respiratory lag is present. This is in marked contrast to the apparent bulging and the exaggerated excursion of the sound side. This has been ascribed by Osler[19] to a condition of vicarious emphysema developing on the unaffected side. The costal margin flares better on the affected side when the lower lobe is the seat of the disorder. This is due to the absence of the antagonistic action of the diaphragm, as described by Hoover.[20]

There is tracheal displacement toward the affected side, most marked in atelectasis of the right upper lobe.

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18. Soltau, A. B.: Brit. M. J. **1**:544 (March) 1925.

19. Osler, W., and McCrae, T.: Principles and Practice of Medicine, New York, D. Appleton & Co., 1923, p. 639.

20. Hoover, C. F.: Am. J. M. Sc. **159**:633 (May) 1920.

Heart: The cardiac impulse is displaced toward the affected side. The superficial area of cardiac dulness may be obscured by the vicarious expansion of the unaffected lung. When the entire lung or either upper lobe is atelectatic, the displacement of the cardiac impulse is obliquely upward, toward the affected side. The cardiac displacement is most marked in cases of right-sided atelectasis.

Lungs: The atelectasis may involve a part of a lobe, an entire lobe or a whole lung. Bilateral involvement may be present and may render the diagnosis almost impossible, because of a lack of appreciable alteration in the negative pressures of the two pleural cavities. When this occurs, the displacement of the mediastinal structures is limited, and the physical signs, dependent on the displacement, do not appear. The most common site is the right lower lobe or the posterior portion of it. The area of atelectasis is triangular, with the base located toward the back. It is interesting to note that this area of the lung is most commonly involved in the atelectasis of premature or weak infants, as described by Holt and Howland.[21]

Dulness, flatness or impaired resonance is present over the atelectatic region. The diaphragm is displaced upward on the affected side and is immobile.

Hirshboeck[22] says that in cases involving the left side the lower part of the chest wall is highly resonant, because of the abnormally high position of the diaphragm. We have found cases in which the liver had been markedly displaced upward, giving rise to tympany over the lower portion of the right wall of the chest.

The vocal and tactile fremitus are diminished in the early stages, and the breath sounds are suppressed or absent. Râles are not present, and the lung in this state has been referred to frequently by many authors as a "dead lung." This is explained by the plugging of the bronchus tributary to the atelectatic area. Increase in the tactile and vocal fremitus, with bronchophony and pectoriloquy, usually appears later and is accompanied by coarse bronchial râles and by mucopurulent expectoration. These changes are especially marked in cases that are complicated by bronchopneumonia. Complete disappearance of all intrathoracic signs usually occurs in from three to four days; in some cases, the signs may remain as long as two weeks, rarely longer.

An elevation in temperature and pulse rate are constantly found in all types of postoperative pulmonary atelectasis. The highest temperature encountered in our series was 106.2 degrees Fahrenheit; the pulse was from 140 to 160 and the respirations from 30 to 54. This

21. Holt, L. E., and Howland, J.: Diseases of Infancy and Childhood, New York, D. Appleton & Co., 1922, pp. 72 and 531.

22. Hirshboeck, F. J. L.: Am. J. M. Sc. **164**:268, 1922.

case was one of general peritonitis. In this connection, it must be remembered that the cases associated with other acute conditions doubtless show a high temperature as a result of the primary pathologic involvement, and the patients suffer an added rise in temperature because of the superimposed burden of atelectasis. In uncomplicated cases, in which simple or interval appendectomies have been performed, with the subsequent development of atelectasis, the temperature ranges between 101 and 103 F., the pulse between 110 and 120, and the respirations between 28 and 35.

A probable explanation of the hyperthermia may be found in the disturbance of equilibrium between the production of heat and the loss of heat. The skin is able to dispose of heat by conduction and radiation and by the evaporation of sweat, depending on the rate of the cutaneous circulation. Through the lung, heat is lost mainly in the vaporization of the water contained in the expired air (MacLeod [14]).

With the patient at rest in bed and covered with blankets, the efficiency of the cutaneous thermal regulating mechanism is decreased; hence, the burden of heat dissipation is thrown on the lungs. Normally, this transfer of function is assumed readily and is adequate; however, in the presence of pulmonary atelectasis, the alveolar surface is diminished and the organism attempts to compensate by increasing the cutaneous circulation; it is unable, however, to effect an equilibrium between production of heat and loss of heat, without a rise in temperature, even in the presence of a compensatory tachypnea.

The pulse rate is increased in proportion to the elevation of temperature, although it has been ascribed to the stimulation of the autonomic nerves and ganglia incident on the displacement of the mediastinal structures. The distress and perturbation of the patient also have been attributed by some to this cause.

The white blood count is not much influenced; it ranges from 8,000 to 12,000 in uncomplicated cases; occasionally it rises to 20,000 in the presence of other associated disorders.

2. *Moderate Type.*—In the moderate type of atelectasis, the symptoms and signs are less severe, and the amount of pulmonary atelectasis is less extensive.

3. *Latent Type.*—The latent cases manifest few symptoms referable to the respiratory tract and are discovered by careful observation and attention to the physical manifestations. These patients usually exhibit a considerable degree of dyspnea when placed on their unaffected side, but not as marked as those with a "frank" or "moderate" type of atelectasis. The physical signs usually clear up in a few days, leaving no residuum; hence, the condition is frequently overlooked. If all patients with slight fever were examined postoperatively as a routine, we believe that more latent cases would be discovered.

4. *Evanescent Type.*—The transient type usually presents moderate or mild symptoms, and its onset is ushered in by a sudden elevation in the temperature, pulse and respiration. The gravity of the symptoms and their duration are subject to marked variation; however, the clinical picture is commensurate with the rise in the temperature, pulse and respiration, in the absence of other complicating pathologic conditions. Simultaneously with the sudden sharp rise of temperature, the patient is extremely perturbed and is apparently the subject of some catastrophe. The symptoms are those presented in a moderate type of atelectasis, and the dyspnea may be extreme. The termination is as abrupt as the onset; after several hours, all the urgent symptoms disappear and the patient is apparently normal. The physical signs disappear coincidently with the clinical improvement. The abatement of the distress is almost invariably associated with the expectoration of thick, tenacious mucus.

Roentgenologic studies in these cases are meager since roentgenograms could not be secured at the proper time.

MODES OF TERMINATION

The condition may terminate in any of the following ways: (1) Crisis, (2) lysis or (3) complication.

In the fifty cases that we studied, thirty-four terminated by lysis; eleven by crisis, and five with complications.

1. *Crisis.*—Termination by crisis entails the rapid reinflation of the atelectatic lung. There may be a profuse expectoration of mucopurulent material or viscid mucus plugs, associated with a clearing of the lung in a few hours. The heart and mediastinal structures assume their normal relations, but the diaphragm on the affected side usually remains immobile for a long period—as much as from one to two weeks—reassuming its function slowly.

2. *Lysis.*—Cases which terminate by lysis present a progressive, gradual abatement of symptoms and signs; coarse bronchial râles, variable in extent and duration, make their appearance, as the mucus plugs are loosened. Eleven patients observed and reported by Elliott and Dingley [4] developed mucopurulent expectoration. Other authors reported the same condition. Over the atelectatic area, there is a gradual return of breath sounds and tactile and vocal fremitus, which may at first be increased and slowly return to the normal type coincident with the removal of the bronchial obstruction. As a rule, this process requires from one to two weeks, but occasionally endures for three.

3. *Complication.*—The course of a case in which a complication develops is that of the complication, which may be either a purulent bronchitis or a bronchopneumonia. The fatal cases are those in which

a bronchopneumonia superimposed on the atelectasis develops. Such patients exhibit an increase in the severity of the symptoms associated with a bloody expectoration and râles over the affected area. These patients, in contrast with those affected merely with an atelectasis, are definitely toxic.

Friction sounds, both pleural and cardiac, have been found in our cases, and have also been reported by others. We believe that in the great majority of the cases, in the absence of pneumonia or bronchopneumonia, the friction sound is caused by the displacement of the thoracic structures and is mechanical rather than pathologic, because it disappears with the return of the structures to their normal position.

Occasionally, pleuritis, with or without effusion, may develop as a late complication.

DIFFERENTIAL DIAGNOSIS

The diagnosis of this condition is relatively easy when the clinical picture is borne in mind. Other conditions and postoperative pulmonary complications, from which differentiation must be made, include the following: pulmonary embolism or infarction; acute dilatation of the heart; pleurisy, with or without effusion; subphrenic abscess; pneumothorax, and diaphragmatic hernia.

Atelectasis is the only condition giving rise to a paradoxic phenomenon. The signs of consolidation of the lung are present, with a suppression of breath sounds and fremitus with retracted, immobile chest, associated with a displacement of the heart and mediastinal structures toward the affected side. In cases in which no complications have occurred, there is an absence of toxicity, and the general symptoms are not so pronounced as in the graver forms of pulmonary complications, such as embolism, infarction or pneumonia. Roentgen-ray examination at the proper time confirms the diagnosis.

Roentgenologic Examination.—The roentgen-ray evidence in pulmonary atelectasis is characteristic and, if obtained at the proper time, will establish the diagnosis. The site of the atelectasis is determined, and the degree of physical displacement and distortion is demonstrated.

In order to cause the least disturbance to the patient, the examination is made with a portable machine. The patient remains in bed, and the roentgenogram is made in the anteroposterior position. The roentgenogram must be examined with special reference to the following points: the line of the vertebrae; convergence of the ribs; position of the heart, aorta and trachea; relative position of the right and left sides of the diaphragm; the presence of increased density in the affected lung and of decreased density in the unaffected lung.

In a typical case of unilateral atelectasis, the following evidence is present:

Vertebrae: The dorsal vertebrae are curved laterally, with the concavity toward the affected side.

Ribs: The ribs converge on the affected side, and diverge on the opposite side, so that one side of the chest space is smaller than normal and the other is larger than normal.

Mediastinum: The heart and mediastinal structures are displaced toward the atelectatic lung. The trachea is displaced less in atelectasis of the left side than in involvement of the right side. The most marked deviation in the trachea occurs with atelectasis of the upper right lobe.

Diaphragm: The diaphragm on the affected side is elevated above its normal relative position and the opposite side of the diaphragm is lower than its normal relative position. In those cases in which the diaphragm has been markedly elevated, fluoroscopic observation shows that slight excursion is present, but so slight that the diaphragm does not play any real part in the respiratory function.

Lungs: On the side of the atelectasis there is increased density, localized or general. This may vary in degree from a slight increase, which is barely perceptible, to a marked increase, great enough to obliterate all markings in the lung and chest. Associated with this, a varying degree of decreased density is found in the opposite lung, the result of the compensatory emphysema.

In bilateral atelectasis, the right and left space in the chest is small. Both sides of the diaphragm are high, but maintain their relative position, and there is no curvature of the spine. There is usually some displacement of the heart toward the more affected side.

There are many cases in which there is no gross density in the affected lung. In the moderate and mild types, there may be present only an inequality of density in the two lungs, increased on the affected side and decreased on the unaffected side, associated with one or several of the following signs: displacement of the heart and mediastinal structures; elevation of the diaphragm, or curvature of the spine, with convergence of the ribs.

Serial roentgenograms are useful in the study of the progress of the condition. As the patient improves, the spine straightens; the ribs assume their normal appearance; the heart and mediastinal structures and the diaphragm gradually return to their normal position, and the densities in the lungs equalize.

It cannot be too strongly emphasized that if one makes the diagnosis of atelectasis only when gross density in the lung is present, many cases will go unrecognized.

The roentgen-ray signs cannot be anticipated by the clinical evidence, nor can the severity of the clinical symptoms be determined by the roentgen-ray evidence. But in general, the more severe the clinical symptoms, the more marked will be the roentgen-ray evidence.

PATHOLOGY

In two cases at autopsy, Jackson and Lee [1] have been able to demonstrate plugs of mucus sufficient to cause complete obstruction to the bronchial lumen and, in vivo, have observed obstructing masses of mucus by means of the bronchoscope. Further bronchoscopic observations by Lee and Tucker [10] have shown an injection of the bronchi as an indication of a mild catarrhal inflammation.

When the chest is opened at autopsy, the lungs do not fill the pleural cavities, and a peculiar swishing sound is produced by the air entering the chest, followed by a return of the retracted wall to its normal position.

Ritvo [23] gives an adequate description of the lung obtained at postmortem: "The involved portion of the lung is blue or violet in color and sharply demarcated from the normal lung tissue. It is soft, tough, and wet; it feels heavy, does not crepitate, and sinks when placed in water. Microscopically, the alveolar surfaces are closely approximated, the alveolar spaces being obliterated." These observations were confirmed at necropsy in one of our cases.

In uncomplicated atelectasis, cellular infiltration is absent, indicating the nonexistence of inflammatory reaction.

TREATMENT

Many observers attempted the amelioration of this condition by the administration of various drugs, especially those producing an effect on the autonomic system, but apparently without benefit. The treatment has therefore been relegated to the symptomatic group, unless a complicating condition arises, in which case the treatment is that of the complication.

Elliott and Dingley [4] advised "keeping the lung open," by encouraging the patients to inspire deeply for five minutes every hour during the first few days after operation, and admonished against the use of tight bandages and straps which restrict the free respiratory act, on the ground that such restriction favors the onset of atelectasis. These authors have employed expectorants and potassium iodide with apparent success. They recommend the production of an artificial pneumothorax on the affected side to relieve the cardiac embarrassment, although they have not used it. Farris [24] has employed this method with apparent benefit in one instance and urges its use in established cases.

An important contribution to the realm of therapy was the institution of bronchoscopic examination coupled with the aspiration of the

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23. Ritvo, M.: Am. J. Roentgenol. **11**:337 (April) 1924.
24. Farris, H. A.: Canad. M. A. J. **15**:808, 1925.

mucus plugs, as detailed in the papers of Jackson and Lee [1] and later, of Tucker [10] and Hearn and Clerf.[16] This method of therapy has recently been employed with success by Harrington.[25]

Manges [15] has proved by roentgen-ray examination that pulmonary aeration is restored following bronchoscopic removal of the obstructing secretion; but repeated aspirations must be carried out, as there is a reaccumulation of mucus in the larger bronchi, followed by recurrence of the atelectasis. This must be performed until such a time as the lung is able to rid itself of the bronchial secretions by the restoration of the cough reflex and increased respiratory force.

SUMMARY

1. Postoperative atelectasis is a definite entity and presents characteristic clinical and roentgenologic evidence, which serves to differentiate it from other postoperative pulmonary complications.

2. Partial atelectasis is the most common type.

3. Prognosis is favorable in uncomplicated cases.

4. Of the fifty cases studied, thirteen were of the fulminant type, seventeen of the moderate type, fifteen of the mild type and five of the evanescent type.

5. Based on our observations, we believe that atelectasis comprises approximately 70 per cent of all postoperative pulmonary complications.

REPORT OF CASES

In the first group in this series, there are twelve cases of the fulminating type.

The last one in this group was not a true postoperative atelectasis, but is of interest because of its infrequency as a postpneumonia complication.

FULMINATING TYPE

CASE 1.—A white woman, aged 22, married, was admitted to the hospital, Dec. 27, 1926. A diagnosis of subacute appendicitis was made, and the patient was operated on the next morning, under nitrous oxide ether-oxygen. The temperature was 99; the pulse, 84, and respirations 22 before operation. The patient was under the anesthetic about thirty-five or forty minutes. Following the operation, she had the usual reaction, except that she complained of mucus in the throat, which was hard to expectorate and caused a constant, hacking cough.

On December 26, she complained of considerable dyspnea and felt as though she were "smothering;" coincidently with this she had profuse diaphoresis, marked brick red, dusky cyanosis, and constant hacking cough. The temperature rose to 103.8. The pulse was 130, respirations, 34. Physical examination at this time showed limitation of movement in the right side of the chest, coarse râles in the upper right side of the chest, dulness in the right base and some displacement of the apex to the right. There was a questionable deviation of the trachea to the

25. Harrington, S. W.: Ann. Surg. **85**:152 (Jan.) 1927.

right; she continued about the same during the night. In the early morning, the nurse reported that she complained of pain on inspiration. The temperature dropped to 101.2; the pulse was 110, and the respirations 28, at noon on December 30. That afternoon the temperature again rose to 104.6. The pulse was 138, the respirations 40, and marked friction rub was present at the base; the breath sounds were absent and the trachea deviated to the right. The condition continued about the same over night; in the early morning a productive cough began, and the patient spat up a large amount of greenish mucopurulent secretion, which relieved her considerably.

The temperature dropped to 100; the pulse to 100, and the respirations to 26 on December 31; the symptoms gradually cleared.

Roentgen-ray examination confirmed the diagnosis.

Case 2.—A white man, aged 22, unmarried, was admitted to the hospital, Nov. 27, 1926. The condition was diagnosed as acute appendicitis. The patient was operated on at 2 p. m., and a ruptured retrocecal appendix was found and removed. The temperature was 101, pulse 100, and respirations 20 at the time of the operation. The patient was placed on Clark's peritonitis treatment immediately. The reaction from the operation brought the temperature up to 101.2, the pulse to 110 and the respirations to 24 at midnight. The following day, he appeared fairly comfortable until the late afternoon, when he complained of increasing dyspnea, profuse diaphoresis and hacking, nonproductive cough. Marked brick red, dusky cyanosis was present. Physical examination at 8:30 p. m. disclosed a marked limitation of movement on the right side, dulness to percussion in the lower right lung and distant breath sounds over this area—apex beat about 3 cm. to left of sternal dine. The trachea was displaced to the right. At 4 a. m., on November 28, the temperature was 104.8; pulse, 126, and respirations, 44. During the early morning, he began having a productive cough and brought up large quantities of mucopurulent secretion, after which he felt somewhat relieved. The temperature had dropped rapidly to 100.8, the pulse to 88, and the respirations to 30 at noon on November 29. The symptoms then gradually subsided, and on December 1, no signs were present in the chest.

Case 3.—A white girl, aged 17, single, was admitted to the hospital, Aug. 18, 1926. The diagnosis was acute suppurative appendicitis, and operation was performed at 6 p. m. under gas induction followed by ether vapor and oxygen; the anesthesia lasted thirty minutes. The abdomen contained free fluid; drainage was instituted and the patient was placed on Clark's peritonitis treatment immediately. The temperature was 102.8; the pulse 136, and respirations 28 at the time of the operation. The temperature was 104.2; the pulse, 140, and respirations 22 at midnight on the day of operation. The next day the temperature was 102.4; pulse, 130, and respirations, 24; the patient apparently was doing well; on the 19th, she complained of much mucus in the throat and coughed up considerable thick tenacious material. That night the nurse turned her on her left side and stepped out for water; she stated that she was not gone more than five or ten minutes; when she returned she found the patient cyanotic, dyspneic and sweating profusely and apparently moribund. The temperature was 105.4; the pulse, 154, and respirations, 54; physical examination was limited to the right side of the chest; the upper right side was literally drowned with coarse, bubbling râles; the breath sounds were absent in the lower right base with dulness to percussion. The apex beat was about 2 cm. from the left sternal border. The trachea was markedly displaced to the right. This condition continued during the night, but the patient felt somewhat improved by noon on August 20. When it was attempted to make the patient comfortable, and she was turned on

the left side, she again became markedly cyanotic and dyspneic. During the day, she spat up large quantities of thick, tenacious, yellowish mucopurulent material. The next morning nothing could be found in the chest. The patient made an uneventful recovery.

Roentgen-ray examination was not made, because the condition cleared up so rapidly.

CASE 4.—A white boy, aged 17, was admitted to the hospital, June 10, 1926. The condition was diagnosed as subacute appendicitis, and operation was performed the same day at 12 p. m., under gas induction, followed by oxygen and ether. The anesthesia lasted one hour and five minutes; a small drain was inserted; the appendix was inflamed but not ruptured.

The patient came out of the anesthetic with a great deal of mucus in the throat and a persistent hacking cough. The temperature caused by the operative reaction was 101.6; the pulse, 92, and respirations, 28 at midnight.

The following day, the patient was still coughing but was expectorating very little: suddenly, about noon, he became cyanotic and dyspneic, and was found lying on his right side, profoundly ill. The temperature was 105.4; the pulse, 30; respirations, 42; from this time until 6 p. m., the patient appeared moribund and perspired profusely; late that night and early in the morning he expectorated a great quantity, from 200 to 300 cc., of thick, purulent, yellowish material; from then on he appeared to improve.

The physical signs were all in the right side of the chest; dulness was present in the lower half of the right lung; a distant breath sound was present; the trachea was displaced; the heart lay 2 cm. from the sternal border in the characteristic attitude.

The patient was thought to be too ill for a roentgen-ray examination, and as the condition cleared up so rapidly, a roentgenogram was not obtained. He made an uneventful recovery.

CASE 5.—M. W., a man, aged 28, was admitted to the hospital, Feb. 10, 1926. The condition was diagnosed as a ruptured appendix, and operation was performed the same day.

The anesthetics used were gas (N_2O), oxygen and ether. The anesthesia lasted 40 minutes. The appendix was gangrenous, retrocecal and ruptured, with free fluid in the abdomen; several gutta percha drains were inserted and Clark's peritonitis treatment was instituted immediately. The postoperative course was stormy for three days, during which the patient complained of hacking, nonproductive cough and some diaphoresis; suddenly in the late afternoon of February 11, he became cyanotic and dyspneic, with irregular shallow breathing, and appeared moribund; there was no distention or other evidence to account for the sudden and severe symptoms; the next morning, no symptoms of importance could be found in chest, but a few râles and some impaired resonance were present in the right side of the chest. The temperature went to 106.4; the pulse to 144, and the respirations to 38, but in the early morning, the respirations were 60 and were irregular and shallow.

On February 13, he again had a severe collapse, with severe dyspnea, cyanosis and profuse diaphoresis; at this time he was found lying on the right side with the head acutely flexed on the right shoulder. The physical signs now were marked; they were: impaired resonance over the lower two thirds of the right side of the chest; the breath sound was absent; the heart was displaced to the right, about 1 cm. from the sternal line; the patient was spitting up great quantities of thick, tenacious mucopurulent material, and complained of severe pain in the right side of the chest. Roentgen-ray examination, Feb. 13, 1926, confirmed the diagnosis.

On February 14, the condition of the chest changed, and it presented the typical signs of bronchopneumonia—almost flatness on percussion; marked impairment of the right side of the chest; increased breath sounds; whispered pectoriloquy and bronchial râles throughout the right side of the chest, but with the displacement of the heart and the mediastinal structures to the right. The patient gradually improved, though with several relapses of cyanosis, dyspnea, etc.

On February 16, he suddenly coughed up a great deal of thick, mucopurulent material and felt much relieved. The final roentgenogram showed evidence of pathologic changes remaining in the lung.

On February 10, the white blood cells numbered 14,600, and on February 20, they numbered 8,900.

On the 18th, the vital capacity of the lungs was —700 cc., and on the 20th, it was —800 cc.

Case 6.—A white man, aged 44, was admitted to the hospital, Jan. 23, 1927. The diagnosis was recurrent right inguinal hernia. The patient was operated on the following day under nitrous oxide induction and ether; he was under the anesthesia one hour and forty minutes. He coughed considerably shortly after he recovered from the anesthetic; it was thought that this man was the type to develop an atelectasis, and therefore he was carefully watched.

The temperature was 99; pulse, 82, and respirations, 18 on the day of the operation; the operative reaction brought the temperature to 100.6, the pulse to 96, and the respirations to 22.

On January 26, the temperature was 102.4; pulse, 130, and respirations, 36; these gradually ascended till on January 27, the temperature was 103.8; pulse, 132, and respirations, 40.

On January 26, physical examination showed: marked cyanosis of the lips and nails, brick red erythema of the face and neck and marked diaphoresis and dyspnea: the right side of the chest showed marked limitation of motion, and even the intercostal spaces could be made out much narrower on the right side; the patient's position also was characteristic; the head was drawn to the right and the thorax inclined to the right; the heart and mediastinal structures were also drawn to the right; dulness was present over the right upper and middle lobe; breath sounds were distant, with absent vocal fremitus and tactile fremitus.

Pleuritic friction rub was present over the upper part of the sternum on the right side; at this time the cough was practically nonproductive and was irritating; the patient complained constantly of "mucus in the throat" that could not be raised.

On January 27, the physical examination was essentially the same, except that coarse râles were now heard, and that the cough was more productive. The patient expectorated large amounts of thick, tenacious mucopurulent material.

On January 30, there were still evidences of pathologic changes in the right lung.

On February 1, the lung was found to be clear, and the roentgenograms were negative (fig. 1).

Case 7.—A white woman, aged 47, was admitted to the hospital, March 3, 1927. The diagnosis was chronic cholecystitis and cholelithiasis; the patient was operated on the following day under gas (N_2O) and ether anesthesia; the operation lasted two hours, as considerable difficulty was encountered in removing the gallbladder. The temperature was 98.8; pulse, 86, and respirations, 20 at the time of the operation; the following morning, the temperature was 104.2; pulse, 126, and respirations, 30.

At noon, on March 4, she began perspiring profusely, was deeply cyanotic and erythematous and was lying with the head toward the right side; the temperature had dropped to 100.6 at noon, but that night it was 104; pulse, 142, and respirations, 36, and the patient appeared moribund.

She began coughing and soon brought up large quantities of thick, tenacious, mucopurulent material.

The condition cleared up gradually, and on March 8, the chest was reported clear.

The condition was confined to the right lower lung; the patient was obese, and physical signs were far from accurate; suppression of the breath sound, dulness over this area and slight deviation of the trachea to the right were present.

CASE 8.—A white boy, aged 16, was admitted to the hospital, Feb. 12, 1927. The diagnosis was acute appendicitis. Operation was performed the same day,

Fig. 1 (case 6).—Appearance of lungs, Jan. 26, 1927, showing involvement of the right upper lobe and marked displacement of the trachea and the mediastinal structures.

under gas (N_2O) and ether anesthesia, and lasted twenty-five minutes. The abdomen contained pus, and the appendix was ruptured; the temperature was 102.4; pulse, 104, and respirations, 34 at the time of operation; the patient was placed on Clark's peritonitis treatment immediately.

That night the temperature was 105.2, and dropped to 102.2 the next morning, but the following day, the patient became cyanotic and dyspneic, began perspiring profusely and seemed moribund; the temperature was 106.6; the pulse, 130, and respirations, 46; the temperature the next day had dropped to 102 again and continued a downward course.

The physical symptoms were definite and typical; the lower half of the right side of the chest was dull with absent tactile and vocal fremitus, and marked displacement of the heart and mediastinal structures to the right. The wall of the

chest showed marked retraction of the intercostal spaces and marked limitation of movement. The lower costal margin moved outward from the midline farther than on the left.

The patient continued to be extremely ill for several days, but about the twelfth or fourteenth day the chest was normal. The roentgenograms were negative.

CASE 9.—A white man, aged 32, was admitted to the hospital, Feb. 22, 1927. The diagnosis was ruptured duodenal ulcer. Operation was immediately performed, and a gangrenous, ruptured appendix was found. The patient was at once placed on Clark's peritonitis treatment. The temperature was 100.6; pulse, 100, and respirations, 20 at the time of operation; that night it receded somewhat. The patient's condition seemed fairly good.

On February 24, the temperature was 104.4; pulse, 144, and respirations, 24. The respiration remained low because of the morphine he was receiving; he became markedly cyanotic and dyspneic and sweated profusely; the pathologic change was limited to the right middle and base of the lung. Distant breath sound was present. Vocal and tactile fremitus were absent.

On February 25, the temperature was 100; pulse, 86, and respirations, 20, and symptoms in the chest were scant.

On February 26, the chest was normal.

CASE 10.—A white man, aged 41, was admitted to the hospital Feb. 22, 1927. The diagnosis was subacute appendicitis, and operation was performed the same day under local field block and light gas (N_2O) and oxygen. The anesthesia lasted for about one hour; from twelve to eighteen hours after the operation he complained of shortness of breath and a tightness through the right side of the chest. The next day he was perspiring profusely, was markedly cyanotic and erythematous, and appeared moribund. The temperature was 100; pulse, 100, and respirations, 22 at the time of the operation; the next day, the temperature was 106; pulse, 146, and respirations, 40. The temperature remained high for two days.

On February 25, the temperature was 100.4; the pulse, 80, and respirations, 20. The entire right side of the chest was practically immobile and the intercostal spaces narrowed. The patient was lying on the right side with the head drawn well over, and the heart and mediastinal structures markedly displaced to the right. He began coughing on February 24, and spit up great quantities of thick tenacious material; the chest showed many coarse rhonchi at this time, and dulness was less marked.

On February 28, the chest was reported clear. The roentgenogram was positive (figs. 2, 3 and 4).

CASE 11.—A white woman, aged 55, was admitted to the hospital, Jan. 7, 1927. Obstipation was present for five days; a palpable mass was present in the upper right quadrant and also a patch of bronchopneumonia in the left base; the patient was extremely ill. Operation was performed early the next day under local block, with 1 per cent procaine hydrochloride; a gangrenous gallbladder was found, with marked adhesions in the hepatic flexure giving symptoms of obstruction; many stones were found in the gallbladder.

The temperature was 99.2; pulse, 100, and respirations, 20 at the time of operation; late that day, the temperature was 103; pulse, 138, and respiration, 24. The patient was gradually growing weaker, but not until January 9 did her condition suddenly become grave; about 5 p. m. she suddenly became cyanotic and dyspneic and the pulse very rapid; profuse diaphoresis was present; the temperature was 105.2; pulse, 144; respiration, 38; there was more definite evidence of pneumonia on the left side, but not as great involvement as would

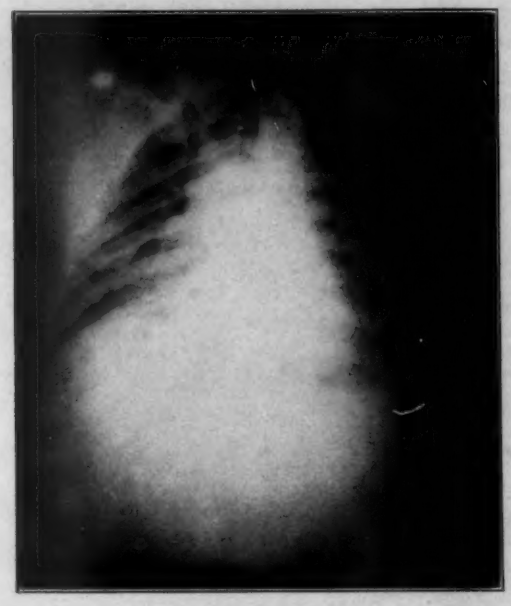

Fig. 2 (case 10).—Appearance, Feb. 23, 1927, showing complete signs of typical atelectasis on the right side and compensatory emphysema on the left side.

Fig. 3 (case 10).—Appearance of lungs on March 11, 1927, showing residual density at the right base. The other signs are normal.

be expected with so sudden a change for the worse; a roentgenogram was made with a portable roentgen-ray apparatus and showed that the right upper lobe was collapsed, with marked deviation of the upper portion of the trachea to the right; she also began coughing and expectorated considerable mucopurulent tenacious material; the temperature was 102; pulse, 120, and respiration, 24 the next morning.

She gradually became weaker and died from toxemia at 9.45 p. m., Jan. 10, 1927. Autopsy was refused.

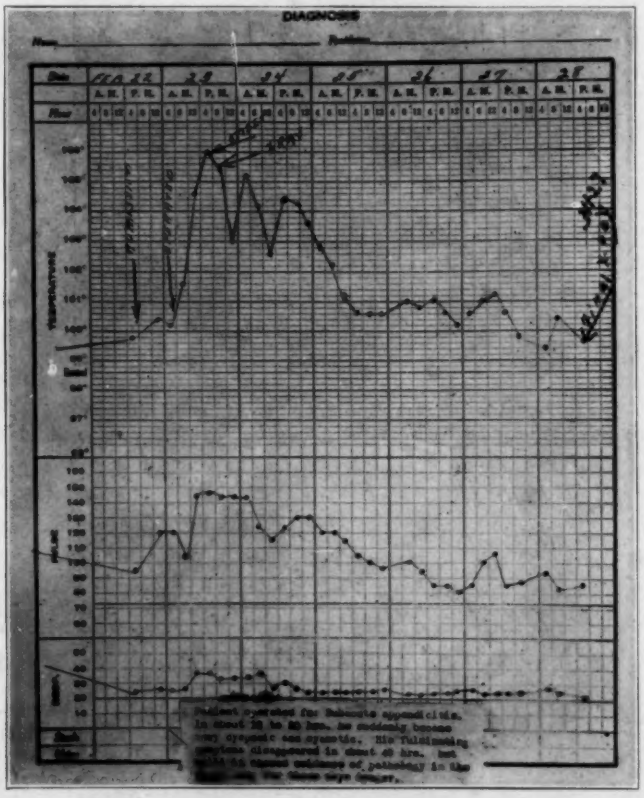

Fig. 4 (case 10).—Summary of course of atelectasis.

CASE 12.—A white woman, aged 42, was admitted to the hospital, May 2, 1926. The diagnosis was subacute cholecystitis. The operation, consisting of cholecystectomy and appendectomy, was performed the following day under drop ether; the anesthesia lasted for one hour and ten minutes. The temperature was 99; pulse, 82, and respirations, 20, at the time of operation. The next day the patient's condition was reported as good. The following morning, she complained of pain in the left side of the chest and marked dyspnea; she became cyanotic, and marked diaphoresis was present. At the same time, a distressing cough began. At 4 p. m., the temperature was 103; pulse, 132, and respirations, 44; dulness over the left lower lung base, a distant breath sound, and displacement of the mediastinal structures to the left were present. In about twelve or

fifteen hours, she began expectorating large quantities of a thick, tenacious, mucopurulent material. The abdomen became greatly distended, and there was a profuse drainage of bile from the incision; she failed rapidly, and died, May 7, of probable peritonitis, acute dilatation of the stomach and myocardial failure. Autopsy was refused. The roentgenogram confirmed the diagnosis of left-sided atelectasis (fig. 5).

CASE 13.—A colored man, aged 28, was admitted to the hospital, Feb. 19, 1926. The diagnosis was made of right lobar pneumonia. The temperature was 104.2; pulse, 120, and respirations, 32; the pneumonia ran an unusual course, which was probably due to the fact that the patient had been given a course of ethylhydrocuprein hydrochloride.

The temperature dropped to normal and remained there for a period of about five or six days, and the patient was convalescing rapidy.

Fig. 5 (case 12).—Appearance of lungs on Jan. 10, 1927, showing atelectasis of the upper right lobe, marked displacement of trachea to the right, pneumonic consolidation in the left lung, marked density in upper right lobe, and decreased density in the middle and lower right lobe, due to compensatory emphysema.

Suddenly, early one morning, he became greatly distressed and was unable to breathe; he perspired profusely and began coughing; on examination, the right side of the chest was dull to percussion, but breath sounds were absent, and the heart was pulled to the right. The patient was thought to have developed an empyema or pleurisy with effusion, but cardiac displacement was reversed. A roentgenogram was then made which showed an atelectasis of the right base.

The condition cleared up in about six or seven days, and the patient apparently recovered, but three days later, he again went through the same symptom complex. The second time it required longer for the symptoms to disappear.

The patient left the hospital with some pathologic change still present in the right base.

This case was not a postoperative one, but is interesting because of its apparent rarity as a complication of pneumonia.

This condition may also be the answer to why many so-called relapses clear up rapidly.

Cases 11 and 12 were really complicated by other conditions which aided in the production of marked elevation of temperature.

The next group of seventeen cases are of the moderate type.

MODERATE TYPE

CASE 14.—A white woman, aged 30, was admitted to the hospital, Jan. 25, 1927. The condition was diagnosed as chronic cholecystitis; cholecystotomy was carried out under gas (N_2O) and ether anesthesia on January 26; the anesthesia lasted forty minutes. The patient recovered from the anesthetic poorly and complained of much mucus in the throat. The temperature was 98.4; pulse, 90, and respirations, 20, at the time of the operation; during the next twenty-eight hours, she complained of tightness in the chest; she sweated profusely and the face was suffused with a dark red blush and some cyanosis of the lips and nails. The temperature was 102.2; pulse, 136, and respiration, 36, on January 28; she began expectorating moderate quantities of tenacious mucopurulent material on the second day. The condition cleared gradually; the chest was clear at the end of five days. The roentgenogram was positive.

CASE 15.—A white woman, aged 25, was admitted to the hospital, Jan. 3, 1927. The diagnosis was retroversion and erosion of the cervix. The patient was operated on the following day under gas (N_2O) and ether anesthesia; the duration of the anesthesia was one hour. At the time of the operation, the temperature was 98.2; pulse, 86, and respiration, 18. The condition was good till about twenty-four hours later, when she began complaining of mucus in the throat and an annoying, nonproductive cough. Coincidently with this she began perspiring profusely and was definitely cyanotic and dyspneic with a marked brick red discoloration of the face and neck. The temperature was 102.6; the pulse, 140, and respiration, 54; dulness was present over the right side of the chest, and there was a distant breath sound in the right base. Coarse râles were present above the dull area; there was limitation of the chest movement on the right side.

On January 5, she began expectorating large amounts of thick, tenacious, mucopurulent material, after which she was considerably relieved.

The condition apparently cleared up, but the patient again developed a cough, which lasted in all about eight days. We thought that she had a purulent bronchitis superimposed on the already existing atelectasis.

CASE 16.—A white man, aged 21, was admitted to the hospital, Jan. 14, 1927. The diagnosis was left inguinal hernia; operation was performed the following day under gas (N_2O) and ether. The duration of the anesthesia was thirty minutes; at the time of the operation, the temperature was 98.4; pulse, 80, and respirations, 20. This patient was annoyed early in the course of the illness by hacking, nonproductive cough and much mucus in the throat.

The following day, in the late afternoon, he complained of pain in the chest, and dyspnea, which are characteristic of this condition. Cyanosis of the lips and nails, brick red erythema of the face and neck, profuse diaphoresis, limitation of motion in the left side of the chest and distant breath sound in the left lower half of the chest were present. The temperature was 102.2; pulse, 112, and respiration, 30; here again the graphic chart belies the apparent clinical

severity. He began expectorating large quantities of thick, tenacious, yellow material and improved rapidly; the chest was clear, January 19. The roentgenograms were positive.

CASE 17.—A white man, aged 39, was admitted to the hospital, Dec. 29, 1926. The condition was diagnosed as chronic cholecystitis and appendicitis.

He was not operated on until December 31, when an appendectomy and cholecystectomy were done. The temperature was 98.4; pulse, 82, and respiration, 20, at the time of operation. The operation was done under gas (N_2O) and ether, lasting one hour and twenty-five minutes; this patient also complained of troublesome mucus in the throat, soon after recovery from the anesthesia. He also complained of cough and pain in the chest.

Twenty hours after the operation, the temperature was 103.4; pulse, 134, and respirations, 38. The patient was perspiring profusely; marked cyanosis of the lips and nails, and the peculiar brick red erythema also were present.

He began expectorating the thick, mucopurulent material rather early, twenty-four hours after the operation and continued for several days. On physical examination, the right side of the chest was found to be limited in movement, dull in the lower right lobe, with distant breath sound and apex beat pulled to the right.

Roentgen-ray examination showed the diaphragm to be high on the right.

The condition gradually cleared up, but the patient left the hospital with slight pathologic changes in the right base.

CASE 18.—A white woman, aged 44, was admitted to the hospital, Feb. 14, 1927. The diagnosis was subacute cholecystitis and cholelithiasis. Operation was performed, February 15, under gas (N_2O) and ether anesthesia; the duration of the anesthesia was forty-five minutes; the patient did badly under the anesthesia, and alpha lobeline, 1/6 grain (0.01 Gm.), was given on the table to stimulate respiration. At the time of the operation, the temperature was 97.6; pulse, 80, and respirations, 20.

That same evening, she began having profuse diaphoresis, and was considerably disturbed by hiccoughs; this lasted for about ten hours. The next morning she began coughing, nonproductively at first, but later she began expectorating a moderate amount of thick, tenacious, mucopurulent material.

On February 16, the temperature was 103.4; pulse, 134, and respirations, 36.

The same morning, she was cyanotic and dyspneic; the reddish erythema was blended with the preexisting icterus; this produced a peculiar, ghastly color, and the patient appeared moribund.

She expectorated considerable thick, tenacious, material and felt somewhat relieved; the condition did not entirely disappear for about eight or nine days.

This patient also had a substernal thyroid, which made the symptoms of dulness in the right side of the chest, absent breath sound and deviation of the trachea to the right somewhat doubtful. However, repeated roentgen-ray examinations made the complications clear.

CASE 19.—The patient was admitted to the hospital, Sept. 11, 1926. The diagnosis was congenital, bilateral indirect inguinal hernia, and an epididymitis. On admission, the patient had a nasopharyngitis and was therefore not operated on until the 14th, when a bilateral herniorrhaphy (Bassini technic) was performed. At the time of the operation, the temperature was 98.2; pulse, 72, and respirations, 20. The operation, performed under gas (N_2O), ether and oxygen, lasted one hour and fifteen minutes. The patient had the usual operative reaction; the temperature was 100.2; pulse, 90, and respirations, 24; the patient apparently was doing well, but had coughed a few times that night; the next day, he appeared

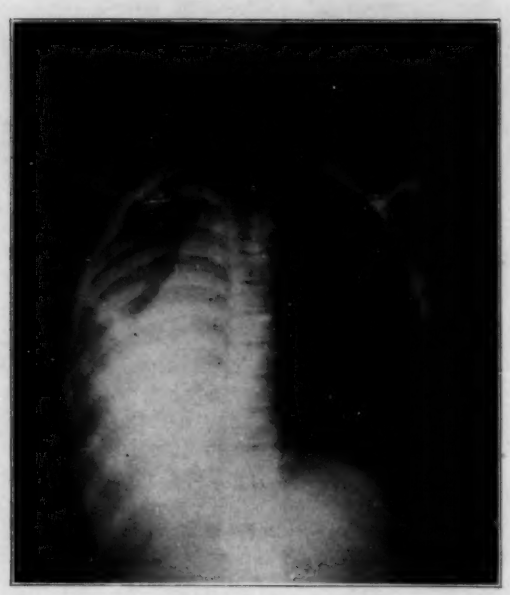

Fig. 6 (case 19).—Appearance of lungs on Sept. 18, 1926, showing atelectasis of the left lung, marked displacement of the heart and aorta to the left, high left side of diaphragm, marked density in the left lung and decreased density in the right lung.

Fig. 7 (case 19).—Normal appearance of lungs on Feb. 23, 1927.

to be improved, but still coughed at infrequent intervals and raised a small amount of yellowish purulent material about 8 p. m.

On September 15, he complained of pain in the left side of the chest and difficulty in breathing; he was perspiring profusely, and the nurse reported him as being very "redfaced." Examination revealed the left side of the chest greatly limited in expansion, dulness in the base of the left lung, with distant breath sound, and almost absent tactile and vocal fremitus; the trachea was somewhat displaced and the apex was in the axillary line. This condition continued through the 16th, and on the 17th he was greatly improved clinically, but still showed

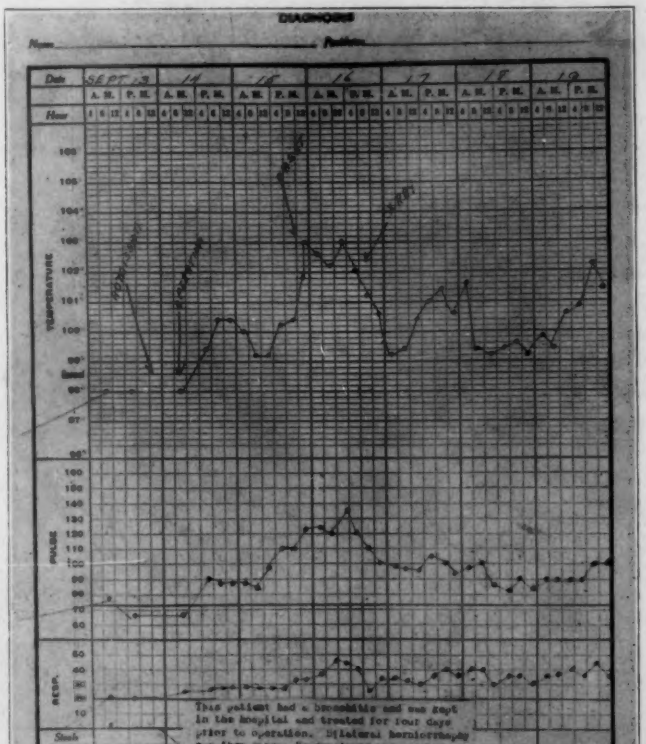

Fig. 8 (case 19).—Summary of course of atelectasis.

physical signs; on the 19th, he had another severe attack similar to the first but it cleared up in about twenty-four hours.

On the 22d, he had entirely returned to normal, clinically as well as physically. Roentgen-ray examination was confirmatory, as shown in figures 6, 7 and 8.

Case 20.—A white girl, aged 12, was admitted to the hospital, Jan. 18, 1927. The diagnosis was subacute appendicitis; operation was performed the following morning under drop ether anesthesia, which lasted twenty-five minutes. The temperature was 99.2; pulse, 110; respirations, 22. The patient progressed favorably, until the evening of the 20th, at 10 p. m., when the nurse reported rising temperature and marked dyspnea. The face was flushed, and the nails and lips slightly

cyanotic; profuse sweating and a hacking, nonproductive cough were present. The temperature was 102.8; pulse, 150, and respirations, 32.

Examination revealed limitation of movement of the left side of the chest, impaired resonance over the left lower lobe, suppressed breath sounds, and apex beat in the axillary line, with very little, if any, displacement of the trachea. The peculiarity of these symptoms was that nearly all of the symptoms in the chest were elicited posteriorly, and very few, if any, could be elicited anteriorly; this is explained by figure 9, which showed the left side of the diaphragm to be higher, the heart and mediastinal structures to the left, and the lung clear; the shadow of the heart was over to the axilla, thus obliterating the patch of atelectasis that must have been behind the heart shadow.

The clinical symptoms had practically disappeared by January 21, but the heart was still displaced, and the diaphragm up when the patient was discharged. She

Fig. 9 (case 20).—Appearance of lungs on Jan. 21, 1927, showing atelectasis of the left lung, displacement of the mediastinal structures to the left, lateral curvature of the spine, with concavity toward the left. The atelectatic area is behind the heart density in the left lung. Increased density in the right lung.

spit up considerable yellow mucopurulent material, which greatly relieved the symptoms.

Case 21.—A white boy, aged 18, was admitted to the hospital, March 17, 1926. The diagnosis of subacute appendicitis was made, and operation was performed the next day under gas (N_2O) induction and ether; the anesthesia lasted for forty minutes. The abdomen was closed without drainage. The temperature before the operation was 98.4; pulse, 78, and respirations, 22. The reaction from the operation caused a temperature of 99.4; pulse, 88, and respirations, 20.

The patient apparently was doing well, when in the early morning of the 19th, he began to cough occasionally; the cough was irritative and nonproductive; the temperature was 101; pulse, 92, and respirations, 28. This condition continued until

the afternoon, when he became markedly cyanotic, dyspneic and began perspiring profusely. The temperature at 9 p. m. was 103.6; pulse, 110, and respirations, 48; the patient was coughing frequently, but the cough was still nonproductive; marked dyspnea, cyanosis and erythema of the face and lips were present. The patient was lying on the right side; dulness over the right base, with diminished breath sound, were present. The trachea and mediastinal structures were displaced to the right apex 3 cm. from the left sternal border.

On March 20, the condition was essentially the same. The next day the temperature and the pulse had subsided to some extent, and the patient felt considerably relieved. The symptoms persisted, with gradual moderation until March 26. The chest still showed some evidence of pathologic change, although clinically the condition was satisfactory.

On March 24, the patient coughed up large amounts of thick, tenacious, mucopurulent material, after which recovery was more rapid.

Case 22.—A colored boy, aged 15, was admitted to the hospital, Dec. 1, 1926. At this time a tonsillectomy was performed, and he was sent home to return later.

The second admission was on Dec. 27, 1927; under drop ether (open mask), an orchidectomy on the left and a Ferguson herniotomy were performed.

At this time, we were considering vagus control in order to determine the etiology of collapse, so the left vagus was vigorously stimulated by rubbing. The temperature was 97.6; pulse, 68, and respirations, 29, at the time of operation. At midnight, the temperature was 98.8; pulse, 100, and respirations, 22.

On December 29, he began coughing slightly and complained of pain in the chest, chiefly on the right side. That afternoon he became dyspneic and perspired a great deal; he was lying in bed with the head and neck inclined to the right. The temperature was 102.8; pulse, 132, and respiration, 38. The lips and finger nails were cyanotic; as the patient was colored, the other erythematous phenomena could not be observed; vocal and tactile fremitus were absent; a distant breath sound was present. The trachea and the mediastinal structures were displaced to the right.

The next day the cough became productive, and he spit up considerable tenacious, mucopurulent material. After this, he felt considerable relief but had numerous coarse râles throughout the upper part of the right side of the chest. The signs gradually disappeared, and on January 3, the lung was found to be entirely clear.

The roentgen-ray evidence was positive.

Case 23.—A white woman, aged 36, was admitted to the hospital, Jan. 25, 1927. The diagnosis was acute appendicitis. Operation was performed under gas (N_2O) and ether; a ruptured appendix was found, with free pus in the abdomen. The anesthesia lasted forty minutes; the appendix was not removed; the abdomen was merely drained.

The following day, the temperature was 106.2; pulse, 124, and respirations, 28; this was an increase from temperature, 103; pulse, 100, and respirations, 18, at the time of operation. The patient was placed on Clark's peritonitis treatment, which kept the respirations lower than would be otherwise expected. She was somewhat dyspneic and cyanotic; an erythematous flush was present; the left side of the chest showed a distant breath sound at the base. Dulness to percussion was present. She developed an ileus and died the following day, Jan. 28, 1927.

The roentgenogram was positive and showed evidence of collapse in the left base.

Case 24.—A white woman, aged 21, was admitted to the hospital, Feb. 23, 1927. The diagnosis was chronic appendicitis; operation was performed the following

day under gas (N_2O) and ether; the anesthesia lasted thirty minutes. In the evening, she complained of mucus in the throat but did not begin coughing until the following day. The temperature was 102.4; pulse, 124, and respirations, 34. In the late afternoon of February 25, the temperature was 97.8; pulse, 90, and respirations, 20, at the time of operation. Physical examination revealed moderate cyanosis; marked diaphoresis, dulness over the lower part of the right lung; distant breath sound and limitation of motion in the right side of the chest and questionable displacement of the apex and mediastinum. The patient coughed up

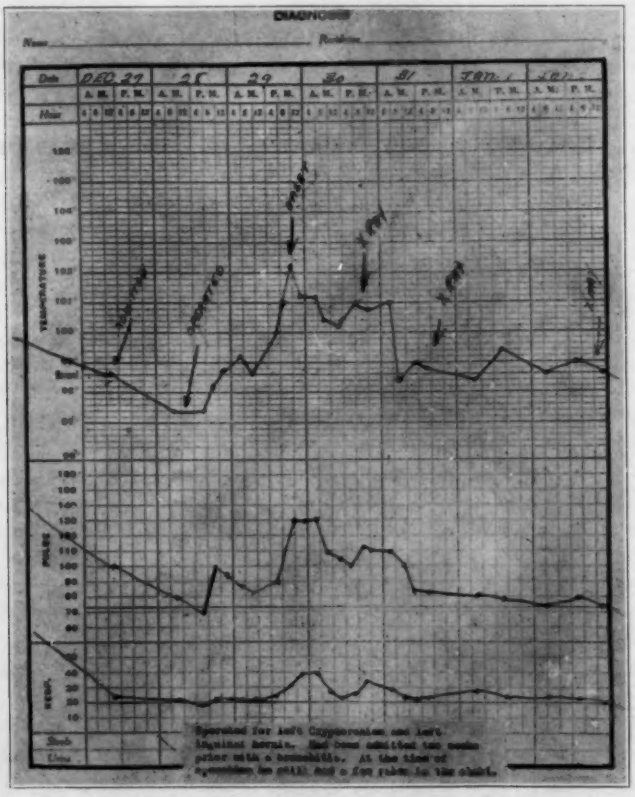

Fig. 10 (case 22).—Summary of course of atelectasis.

a moderate amount of thick, tenacious, mucopurulent material on the 26th, and the chest was entirely clear on the 28th.

Case 25.—A white woman, aged 35, was admitted to the hospital, Feb. 21, 1927. The diagnosis was retroversion, 3 degrees. Stenosis of the cervix and hemorrhoids were found. Operation was performed the following day under gas (N_2O) and ether; the duration of the anesthesia was one hour and five minutes. The temperature was 98; pulse, 84, and respirations, 18, at the time of operation. The patient complained of considerable mucus in the throat on awakening from the anesthesia. In the early morning of the 23d, she began coughing and complained of pain in the left side of the chest; she began perspiring freely; the night nurse reported the patient as "very red-faced and having trouble breathing."

The upper quadrant of the left side of the chest was dull, and a distant breath sound, limited movement and marked erythematous flush were present. The patient began coughing at this time; the temperature was 101.6; pulse, 114, and respirations, 28. She spit up considerable mucopurulent material; this condition of the chest disappeared in about forty-eight hours.

CASE 26.—A white man, aged 40, was admitted to the hospital, Feb. 9, 1927. The diagnosis was bilateral indirect hernia. Operation was performed on February 11, 1 per cent procaine being used for field block. The patient had a nasopharyngitis and was coughing occasionally. The temperature was 97.4; pulse, 68, and respirations, 18, at the time of operation. He continued coughing and the next day began perspiring profusely and became dyspneic; brick red erythema appeared on the face and neck. The temperature was 101.6; pulse, 114, and respirations, 38; in the late afternoon of the 12th, he presented the following symptoms: nonproductive coughing; a feeling of tightness in the chest and marked cyanosis and erythema when he lay on the right side. There was dulness to percussion over the right side, distant bandbox sound and marked limitation of motion in the right side of the chest. The intercostal spaces were markedly retracted. He expectorated considerable purulent tenacious material, but continued to present symptoms for six or seven days. The chest was reported as normal on the 18th.

The roentgen-ray examination was positive.

CASE 27.—A white man, aged 68, was admitted to the hospital, Feb. 9, 1927. The diagnosis was appendical abscess.

On February 14, operation was performed under spinal anesthesia, 10 cc. of 1 per cent procaine hydrochloride being injected into the third lumbar space; the operation was completed in thirty-five minutes; the appendix was removed and the abscess drained. The temperature was 98.4; pulse, 84, and respirations, 20, at the time of operation. This patient also had a valvulitis, the mitral valve being stenosed and incompetent; he suffered no reaction from the spinal anesthesia.

On February 17, the patient suddenly complained of tightness in the chest and dyspnea; it was noticed that he became markedly cyanotic and erythematous, and almost immediately covered with perspiration. Physical examination revealed dulness in the lower quadrant of the right side of the chest, distant breath sound and displacement of the heart and mediastinum to the right; the patient also complained of chill, this being the first time we had encountered this symptom.

The condition continued about the same for two days, and seemed to have cleared up. Then the patient had a severe chill, and sweating began again; the left side was found to contain evidence of patchy bronchopneumonia; he gradually failed and expired, Feb. 21, 1927. The temperature was 102.4; pulse, 116, and respirations, 40, on the 17th. The temperature returned to 100.1 on the 19th; then it shot up again on the 20th, and continued upward to the end.

The roentgen-ray was positive for atelectasis in the right base and later for a confluent bronchopneumonia in the left base.

CASE 28.—A white man, aged 61, was admitted to the hospital, Feb. 13, 1927. The diagnosis was right indirect inguinal hernia. Operation was performed the following day under gas (N_2O) and ether; the duration of the anesthesia was one hour. The temperature was 98.4; pulse, 70, and respirations, 18, at the time of operation; the patient had coughed a few times prior to the operation. February 15, the temperature was 101.6; pulse, 96, and respirations, 28, and he began to complain of marked difficulty in breathing, and became somewhat cyanotic. Erythema of face and neck and moderate diaphoresis were present, but he began spitting up considerable thick, mucopurulent material. The pathologic

change was limited to the right lower lung; diminished breath sound, dulness to percussion and moderate displacement of the heart and mediastinal structures to the right were present.

On February 18, the chest was reported normal; in the interim, he expectorated much tenacious, yellow mucopurulent material. Roentgen-ray examination was positive for atelectasis in the right base.

CASE 29.—A white boy, aged 18, was admitted to the hospital, Feb. 23, 1927. The diagnosis was bilateral indirect inguinal hernias. Operation was performed the following day under spinal anesthesia, 10 cc. of 1 per cent procaine hydrochloride being injected in the second lumbar interspace; this form of anesthesia was used because the patient had a bronchitis. The temperature was 97.6; pulse, 70, and respirations, 18, at the time of operation; there was no reaction from the spinal anesthesia; the patient was troubled during that night with a hacking cough, but was no worse than on admission. Early the following morning, he became definitely cyanotic and dyspneic, with marked diaphoresis. The temperature was 101.2; pulse, 110, and respirations, 36. The cough became productive; examination at this time showed some dulness over both lower lobes, but most marked over the right; distant breath sound and some limitation in movement of the wall of the chest. The heart and mediastinum were somewhat displaced to the right. The patient began spitting up large quantities of mucopurulent material, and the condition cleared up rapidly, no evidence of pathologic change being found in the chest on February 27, except a few coarse râles, which were inconstant.

CASE 30.—A white man, aged 40, was admitted to the hospital Feb. 1, 1927. The diagnosis was chronic duodenal ulcer. Posterior gastro-enterostomy was done, Feb. 7, 1927, under gas (N_2O) and ether; the duration of the anesthesia was two hours and fifteen minutes. Considerable difficulty was experienced in mobilizing the jejunum.

At the time of the operation, the temperature was 97.6; pulse, 70, and respirations, 20. The following day, the temperature was 102.4; pulse, 106, and respirations, 28; the patient was moderately cyanotic, but was quite dyspneic, and had marked facial erythema. He appeared much sicker than the temperature chart showed.

He was lying with the head inclined to the right. Examination revealed marked limitation of motion in the right side of the chest, distant breath sound over the right base, dulness on percussion and moderate displacement of the apex to the right. The following day he began coughing and expectorating moderate amounts of thick, tenacious, yellow material; the condition in the chest improved, but rhonchi were present.

On February 11, the chest was found to be clear. The roentgenogram showed evidence of atelectasis in the right base.

MILD TYPE

CASE 31.—A white woman, aged 24, was admitted to the hospital, Jan. 10, 1927. The diagnosis was bilateral salpingitis. The patient was operated on the following day under gas (N_2O) and ether. The duration of the anesthesia was one hour. The same evening, the temperature was 102.2; pulse, 124, and respirations, 30, in contrast with a temperature of 98.4; pulse, 90, and respirations, 20, at the time of operation. She perspired profusely and complained of mucus in the throat and marked erythema of the face. The temperature dropped suddenly the next day, and was normal at 4 p. m., January 12.

She spit up considerable mucopurulent, tenacious material and felt much relieved. Dulness in the lower part of the right lung, distant breath sound

over this area, questionable displacement of the mediastinal structures and a few coarse râles in the upper part of the right side of the chest were found. She continued to spit up a moderate amount of this material for three days. The clinical syndrome disappeared in about thirty-six hours.

CASE 32.—A white man, aged 27, was admitted to the hospital, Jan. 1, 1927. The diagnosis was subacute appendicitis. Operation was performed the following day. The wound was clean, so no drainage was used. The anesthetic used was gas (N_2O) and ether, and the operation lasted fifty minutes. Within eight hours after the operation, he complained of cough and mucus in the throat, and marked brick-red erythema of the face and neck; the next morning, he was spitting up great quantities of thick, tenacious, mucopurulent material; it was thought that he had an acute abscess of the lung from the amount of the material expectorated. The temperature was 101.2; pulse, 120, and respirations, 36. His distress was much more marked than was evidenced by the graphic chart.

On January 4, he was still spitting up large amounts.

On the 5th, he was comfortable; the temperature was 98.4; pulse, 92, and respirations, 20, the condition having cleared up practically over night. A roentgenogram taken late on the 4th was reported normal; an earlier picture was missed.

CASE 33.—A white woman, aged 34, was admitted to the hospital, Feb. 13, 1927. The diagnosis was chronic appendicitis and femoral hernia.

Appendectomy and herniorrhaphy (femoral) were performed the following day under gas (N_2O) and oxygen. The duration of the anesthesia was one hour.

At the time of the operation the temperature was 98.6; pulse, 84, and respiration, 20. The same evening the patient complained of mucus in the throat and was perspiring moderately. The following day she had the characteristic reddish erythema of the face and neck and was somewhat cyanotic and dyspneic. The temperature was 101.8; pulse, 110, and respirations, 30. Soon after this, she began coughing and expectorating a moderate amount of thick, tenacious, mucopurulent material. Physical examination on February 15 showed: dulness in the left side of the chest, upper portion; the apex displaced upward and somewhat to the left and profuse diaphoresis. Distant breath sound was elicited over the left side of the chest.

The condition gradually subsided, and the chest was clear, February 19.

The roentgenogram was positive (fig. 11).

CASE 34.—A white woman, aged 66, was admitted to the hospital, Jan. 30, 1927. The diagnosis was prolapse of the uterus, umbilical hernia, enlarged thyroid and moderate degree of bronchitis.

The patient was operated on the following day, under local field block with 1 per cent procaine hydrochloride; umbilical herniorrhaphy was done.

The following day, in the early afternoon, she complained of some pain in the right side of the chest; during the night she began sweating profusely and developed an annoying cough. The temperature was 101.6; pulse, 118, and respiration, 30, at 4 a. m. on February 1.

On physical examination the next morning at 9 a. m., she showed some cyanosis and moderate dyspneic and erythematous suffusion of the face and neck. The patient was large and heavy, and physical examination was unsatisfactory, but because of a previous experience with this symptom complex, roentgen-ray examination was ordered.

A substernal thyroid had displaced the trachea to the right, but we felt that the pathologic change in the right upper lobe was atelectasis, so a roentgenogram was made, which showed a partial return of the trachea to its normal position.

The condition cleared up by lysis, and the chest was clear, February 6.

Case 35.—A white man, aged 36, was admitted to the hospital, Jan. 17, 1927. The diagnosis was subacute appendicitis. Operation was performed the following morning under gas (N_2O) and ether. The anesthesia lasted forty-five minutes. Some difficulty was experienced in removing the appendix, as it was firmly bound to the cecum by adhesions. The temperature was 98; pulse, 80, and respirations, 20, at the time of operation. The temperature reached 100 that night; the pulse, 86, and respirations, 20. Soon after the operation, the patient began hiccoughing, which persisted for about twenty-eight hours and which was finally checked by the old-fashioned expedient of holding out the tongue for five or ten minutes.

On January 19, in the morning, he began complaining of severe pain in the chest and began coughing; the cough was nonproductive, but very distressing. At this time he also became cyanotic and a brick red erythema of the face and

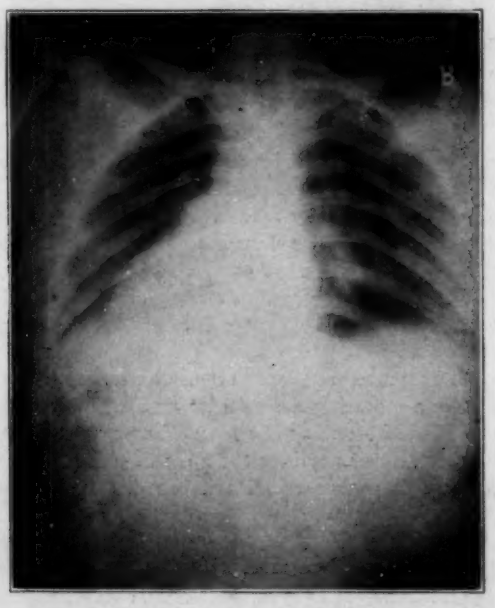

Fig. 11 (case 33).—Appearance of lungs on Feb. 17, 1927, showing involvement of the left lung, spine curved laterally, with concavity toward the left. The left ribs converge, and the right ribs diverge. The heart is displaced to the left. The left side of the diaphragm is elevated. Increased density in the left base.

hands appeared. The diaphoresis became so profuse that a change of bed clothing was necessitated four or five times daily. Physical examination disclosed marked displacement of the mediastinum to the right, diminished breath sound over the right lung, a few râles and marked pleuropericardial friction rub. There was a marked asymmetry between the right and left sides of the chest. Inspection showed an immobilization of the right side of the chest. A peculiar condition was found on percussion. A "strap-like" area of dulness was found extending from the fourth to the sixth interspace on the right side of the chest anteriorily, and below this a booming resonance could be heard. This was explained by the roentgenogram taken shortly after, which showed that the diaphragm had been displaced upward so far that a great dome had been formed

Fig. 12 (case 35).—Appearance of lungs on Jan. 19, 1927, showing atelectasis of the right lung, especially at the base. The mediastinal structures are displaced to the right. Marked displacement of the right side of the diaphragm is present. The liver is displaced up with the diaphragm so that its lower border is almost on the same level with the left side of the diaphragm. Marked density in the base of the right lung, general increased density in the rest of the right lung and decreased density in the left lung are present.

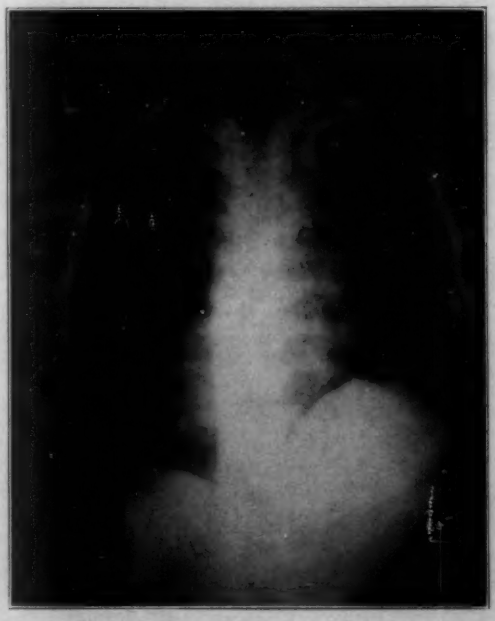

Fig. 13 (case 35).—Appearance on Jan. 22, 1927, showing less displacement of the mediastinal structures, the density disappearing from the right lung, and the right side of the diaphragm still elevated. Fluoroscopic examination at this time showed slight excursion in the right side of the diaphragm.

which had pulled the liver along, thus eliminating the usual liver dulness. The condition in the chest persisted for several days and abated by lysis. The highest temperature was 101.6; pulse, 120, and respirations, 26. This was not in keeping with the condition as shown by the roentgenogram and by the marked physical characteristics.

The roentgenographic appearance in this case is shown in figures 12 and 13. The vital capacity of the lungs was 1,500 cc. The white blood cells numbered 8,500 on January 20.

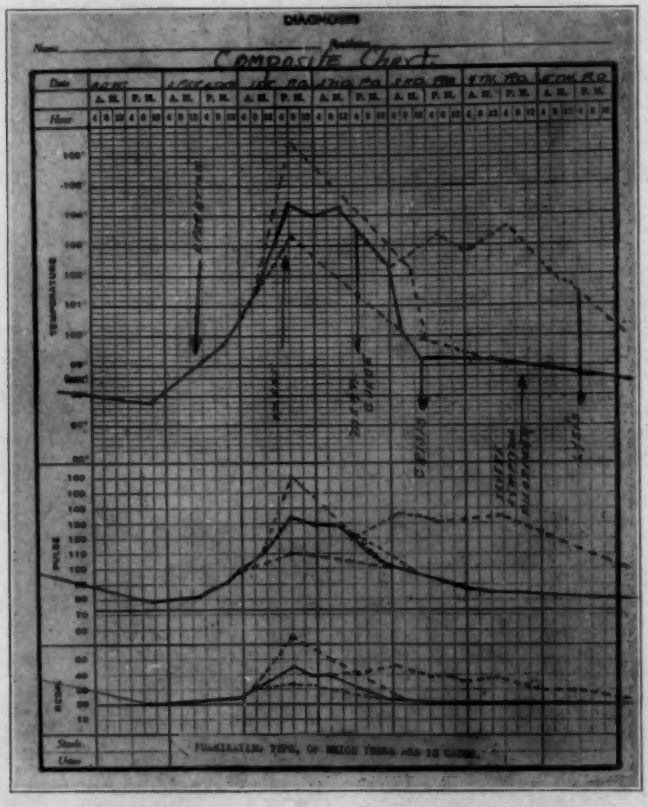

Fig. 14.—Composite chart of thirteen cases of the fulminating type of atelectasis.

On January 22, the patient stated that he felt as though something had snapped in the chest; he spit up a large amount of mucopurulent material, and from then on experienced great relief and has felt no discomfort since.

Case 36.—A white woman, aged 36, was admitted to the hospital, Nov. 30, 1926. The diagnosis lay between ruptured appendix and pelvic peritonitis. Operation was performed the same day under gas (N_2O) and ether; a ruptured appendix was found with pus in the abdomen, and involving the right tube and ovary. The abdomen was drained in three places, and the patient was placed on Clark's peritonitis treatment; she was in a critical condition for about twelve days; she developed a phlebitis in the right leg on December 9.

The temperature was 101.6; pulse, 120, and respirations, 26, at the time of operation. A temperature of 105.4, pulse of 140, and respirations of 24, on December 2, gradually subsided to a temperature of 100.2, a pulse of 98, and respiration of 20 on December 8.

On the 10th the patient developed marked dyspnea, cyanosis and pain in the right side of the chest and began coughing. At this time, the temperature was 101.8; pulse, 110, and respiration, 36. On examination, the lower part of the right side of the chest was found to be limited in movement, with dulness to percussion; a distant breath sound, but no egophony or pectoriloquy was

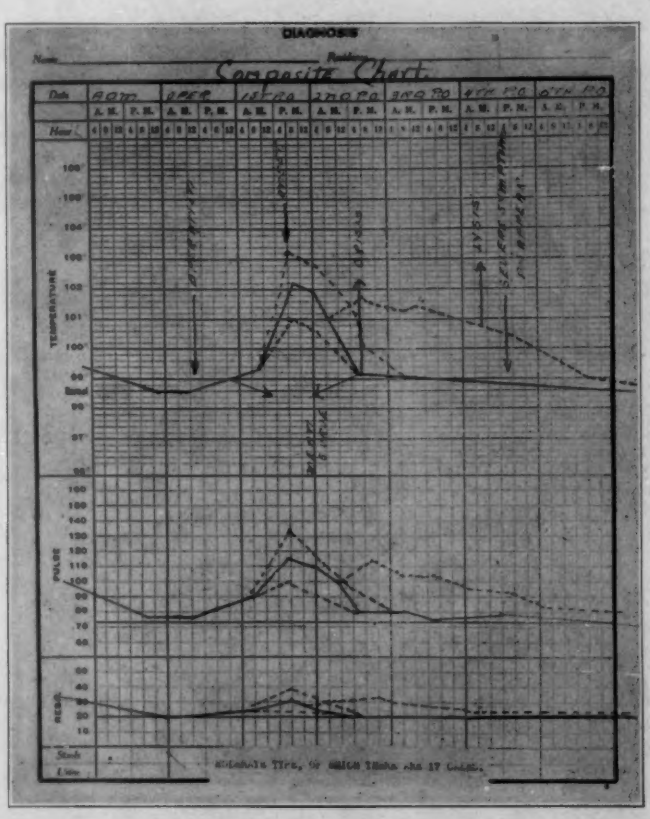

Fig. 15.—Composite chart of seventeen cases of moderate type of atelectasis.

present; the mediastinal structures were pulled to the right; after ten hours a productive cough began and during the next few hours she spit up a large quantity of thick, mucopurulent, tenacious material. The chest was clear at the end of forty-eight hours, and the patient went on to an uneventful recovery.

CASE 37.—A white man, aged 34, was admitted to the hospital, Feb. 9, 1927. Bilateral herniorrhaphy was performed, February 10, under gas (N_2O) and ether; the duration of the anesthesia was one hour and fifty minutes. The day of the operation the temperature was 98.4; pulse, 72, and respirations, 18.

The following day, in the morning, he complained of considerable pain in the left side of the chest; no cough, diaphoresis or dyspnea were present. During

the evening and early morning, he became somewhat cyanotic; brick red erythema was present, and he complained of dyspnea. He began coughing at this time.

On February 12, the following physical signs were present: the patient was found lying on the left side, with the head averted; dulness and no breath sound were present over the lower part of the left lung, as well as inspiratory limitation of the left side of the chest; the patient was now cyanotic, and was perspiring freely.

Marked dyspnea was found when the patient was turned to the unaffected right side; only slight displacement could be made out.

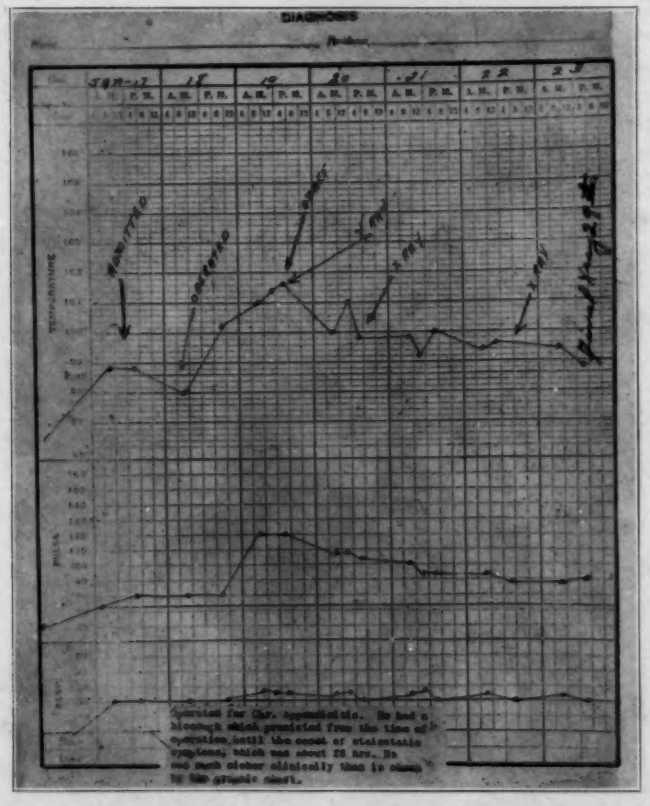

Fig. 16 (case 35).—Summary of course of atelectasis.

On February 13, he coughed up several mucus plugs, after which he felt greatly relieved. The chest was found to be entirely clear on February 17.

The roentgenograms were positive.

Case 38.—A white man, aged 52, was admitted to the hospital, Feb. 3, 1927. A bilateral herniorrhaphy was performed, February 9, under gas (N_2O) and ether; the anesthesia lasted one hour and ten minutes. The temperature was 99; pulse, 76, and respirations, 20, on the day of the operation. During the day, after the recovery from the anesthetic, he complained of considerable mucus in the throat; on February 10, the patient complained of pain in the chest and an annoying, nonproductive cough. On examination, he was found to be perspiring freely, was somewhat cyanotic and was lying on the right side. Breath sounds

were diminished over the right base; dulness on percussion was found over this area, and there was only slight displacement of the heart and mediastinum to the right, he also had the typical brick red discoloration of the face and upper side of the chest and neck; shortly after, he began expectorating large amounts of thick, mucopurulent material. The temperature was 101.2; pulse, 94, and respirations, 26. After coughing and expectorating freely, the patient felt much relieved.

On February 12, the condition was found to be clear. The roentgenogram was positive.

CASE 39.—A white man, aged 21, was admitted to the hospital, Jan. 25, 1927. The diagnosis was chronic appendicitis. Operation was performed the next day under gas (N_2O) and ether; the duration of the anesthesia was forty minutes. The appendix was removed, but no drain was inserted. The temperature was 98; pulse, 74, and respirations, 18, at the time of the operation; that afternoon, the temperature was 99.6; pulse, 90, and respirations, 22, due to the operation. Early the next day the patient had a nonproductive cough of an annoying type and complained of mucus in the throat, which he was unable to raise. The temperature was 100.4; pulse, 88, and respirations, 26. Although the temperature, etc., was not greatly increased, he nevertheless was somewhat cyanotic and perspired freely; there was also some displacement of the mediastinal structures; slight evidence of displacement of the trachea and a dull area over the lower part of the left lung with distant breath sign. The physical signs were not marked. The roentgenogram was positive for atelectasis.

CASE 40.—A white woman, aged 29, was admitted to the hospital, Jan. 19, 1927. The diagnosis was acute cholecystitis. Operation was performed the following day under gas (N_2O) and ether. The gallbladder was found gangrenous and consequently was merely drained. The patient made a good recovery and was operated on again on January 28. At this time a cholecystectomy was performed under gas (N_2O) and ether. The following day the patient began perspiring profusely and complained of pain in the right side of the chest. She was moderately cyanotic and erythematous. The temperature was 99; pulse, 94, and respirations, 22, at the time of the operation. The following day the temperature was 101.8; pulse, 14, and respirations, 38. A few hours later she began coughing and spit up considerable thick, tenacious, mucopurulent material, after which she made an uneventful recovery. The pathologic change was limited to the right base, over which area the breath sounds were distant, and impairment to percussion was present.

CASE 41.—A white woman, aged 22, was admitted to the hospital, Feb. 11, 1927. The diagnosis was chronic appendicitis. Operation was performed the following day under gas (N_2O) and ether. The duration of the anesthesia was one hour and ten minutes. Considerable difficulty was encountered, as the appendix was retroperitoneal. No drain was used. Early the following morning, she complained of a choking sensation and much mucus in the throat; she became somewhat cyanotic and markedly erythematous about the face and neck with considerable diaphoresis. She began coughing early and spit up considerable thick, tenacious mucus. The temperature was 97.8; pulse, 84, and respirations, 20, on the day of the operation. The following day, the temperature was 100.8; pulse, 116, and respirations, 30. The symptoms were localized in the right base; some dulness was present, and the breath sound was absent. The chest was clear in about forty-eight hours.

CASE 42.—A white man, aged 22, was admitted to the hospital, Feb. 20, 1927. The diagnosis was bilateral inguinal hernia. Operation was performed the

following day under gas (N_2O) and ether. The duration of the anesthesia was one hour and five minutes. The temperature was 98.4; pulse, 72, and respirations, 18, at the time of the operation; about twenty hours later the patient became moderately cyanotic and erythematous, with some diaphoresis. There was some evidence of pathologic change in the right base; distant breath sound and some impairment on percussion were present. These signs practically subsided in about forty-eight hours. The temperature was 100; pulse, 104, and respirations, 28, on February 22. The symptoms had disappeared clinically by the following day.

Case 43.—A white woman, aged 66, was admitted to the hospital, Feb. 8, 1927. The diagnosis was cholecystitis and cholelithiasis. Operation was performed, February 21, under gas (N_2O) and ether. The duration of the anesthesia was one hour and twenty minutes.

The temperature was 97.4; pulse, 86, and respirations, 22, at the time of the operation. The following day the patient was markedly cyanotic and erythematous and complained of considerable pain in the left side of the chest and difficulty in breathing; the left base was found to be impaired in movement and dull to percussion; distant breath sound and apex beat were present in the axillary line. The temperature was 101.2; pulse, 112, and respirations, 28. She began coughing shortly and spit up a thick, tenacious material rather copiously; the condition gradually cleared up, and the chest was normal five days later.

Case 44.—A white man, aged 56, was admitted to the hospital, Feb. 10, 1927. The diagnosis was chronic cholecystitis and cholelithiasis. The patient was operated on February 17, under gas (N_2O) and ether; the duration of the anesthesia was one hour and fifteen minutes. At the time of the operation the temperature was 98.6; pulse, 76, and respirations, 20. Late the following afternoon the temperature was 100.8; pulse, 120, and respirations, 30, and the patient was definitely cyanotic and perspiring freely, with brick-red erythema of the face and neck. On examination, a definite dulness was found over the right base, especially posteriorly. There was diminished excursion of the right side of the chest. A roentgenogram taken, February 18, showed some displacement of the mediastinal structures. A roentgenogram taken the next day showed more evidence of collapse on the right side but also showed a patch of bronchopneumonia on the left. The final examination showed an almost normal chest. The condition cleared up rapidly, and on February 21, the chest was reported clear.

Case 45.—A white man, aged 38, was admitted to the hospital, Nov. 25, 1926. Operation was performed on December 10 under drop ether; the anesthesia lasted for one hour and forty-five minutes; bilateral herniorrhaphy was done, and left indirect inguinal hernia, and recurrent inguinal hernia on the right were found. The temperature was 98.2; pulse, 76, and respirations, 18, at the time of the operation.

In the late afternoon of the 11th, he became cyanotic and dyspneic, although the respiratory rate was not greatly increased; he had a persistent irritating, nonproductive cough. Physical examination revealed dulness in the right base, numerous râles above this area, no breath sound over the right base and marked limitation of movement of the right side of the chest. This condition persisted for about thirty-six hours, during which he spit up copious amounts of mucopurulent material; the chest had returned to normal in about forty-eight hours, although he had an irritating cough for about twenty-four hours longer. The fastigium showed a temperature of 101.4; pulse, 94, and respirations, 26.

The patient made an uneventful recovery from the 14th to the time of discharge.

The following five cases were of the evanescent type.

EVANESCENT TYPE

CASE 46.—A white man, aged 24, was admitted to the hospital, Feb. 7, 1927. The diagnosis was bilateral inguinal hernia. Operation was performed the following day under gas (N_2O) and ether; the duration of the anesthesia was one hour and five minutes. The temperature was 96.4; pulse, 86, and respirations, 20, on the day of the operation. About twenty-six or twenty-eight hours later, he was moderately cyanotic, with some erythema of the face and neck and moderate diaphoresis; the cough was almost immediately productive, and he spit up considerable yellowish, tenacious material. The attack began suddenly, with pain

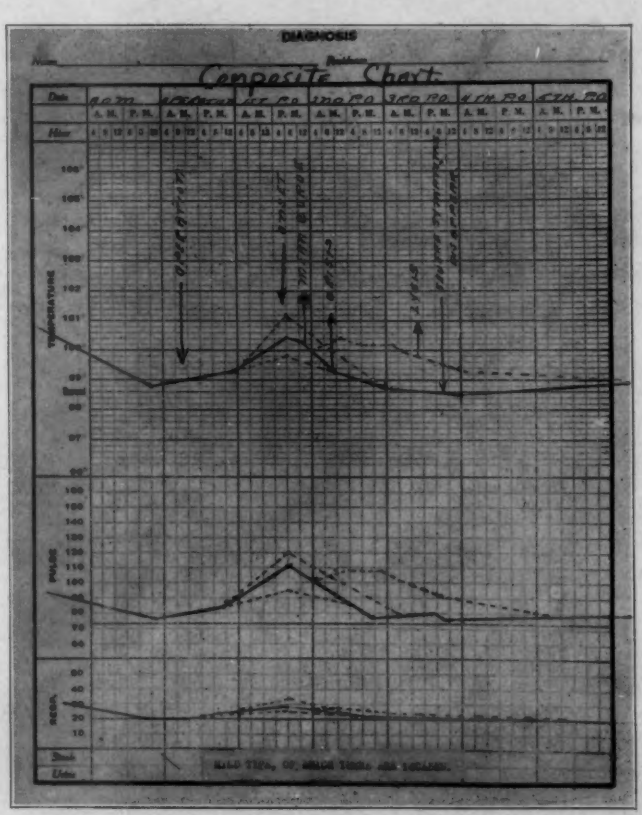

Fig. 17.—Composite chart of fourteen cases of the mild type of atelectasis.

in the upper part of the left side of the chest, in which a slight dulness on percussion and distant breath sound were present. About twenty-four hours later, all signs had disappeared. The temperature was 101; pulse, 100, and respirations, 26, on February 9.

CASE 47.—A white man, aged 46, was admitted to the hospital, Feb. 6, 1927. The diagnosis was bilateral indirect inguinal hernia. The patient was operated on the following day, and bilateral Bassini herniorrhaphy was done. The anesthesia lasted fifty-five minutes. Gas (N_2O) and ether were given. The temperature was 98.4; pulse, 84, and respirations, 20, at the time of the operation. The patient was apparently making a good recovery until the early morning of

TABLE 1.—*Summary of Fifty Case Histories*

Case	Age	Type	Operations	Onset	Regression
1	17	Fulminant	Appendectomy	26 hours	Crisis
2	22	Fulminant	Appendectomy	26 hours	Lysis
3	17	Fulminant	Appendectomy	30 hours	Crisis
4	28	Fulminant	Cholecystectomy	64 hours	Complication; peritonitis; hepatitis
5	55	Fulminant	Cholecystectomy	30 hours	Complication: lobar pneumonia
6	22—	Fulminant	Appendectomy	32 hours	Lysis
7	28	Fulminant	Appendectomy	20 hours	Lysis
8	28	Fulminant	Perineorrhaphy	10 days	Lysis
9	44	Fulminant	Herniorrhaphy	20 hours	Lysis
10	16	Fulminant	Appendectomy	18 hours	Lysis
11	12	Moderate	Appendectomy	30 hours	Crisis
12	21	Moderate	Herniorrhaphy	30 hours	Crisis
13	13	Moderate	Herniorrhaphy	36 hours	Lysis
14	15	Moderate	Herniorrhaphy; orchidectomy	36 hours	Lysis
15	28	Moderate	Appendectomy	28 hours	Lysis
16	39	Moderate	Appendectomy	20 hours	Lysis
17	36	Moderate	Appendectomy	28 hours	Complication: peritonitis
18	40	Moderate	Herniorrhaphy (bilateral)	24 hours	Lysis
19	61	Moderate	Herniorrhaphy (right)	30 hours	Lysis
20	68	Moderate	Appendectomy	72 hours	Lysis
21	40	Moderate	Gastro-enterostomy	28 hours	Lysis
22	44	Moderate	Cholecystectomy	26 hours	Lysis
23	25	Moderate	Appendectomy; suspension	24 hours	Lysis; bronchitis
24	30	Moderate	Cholecystotomy	30 hours	Lysis
25	36	Mild	Appendectomy	26 hours	Crisis
26	38	Mild	Herniorrhaphy	28 hours	Lysis
27	42	Mild	Appendectomy	10 days	Lysis
28	21	Moderate	Appendectomy	28 hours	Lysis
29	52	Mild	Herniorrhaphy (right)	26 hours	Crisis
30	34	Mild	Herniorrhaphy (bilateral)	24 hours	Lysis
31	66	Mild	Herniorrhaphy	24 hours	Lysis
32	34	Mild	Perineorrhaphy; herniorrhaphy (femora)	30 hours	Lysis
33	22	Mild	Appendectomy	24 hours	Lysis
34	29—	Mild	Cholecystectomy	18 hours	Lysis
35	24	Mild	Salpigectomy	14 hours	Lysis; complication: bronchitis
36	27	Mild	Appendectomy	26 hours	Lysis
37	55	Mild	Cholecystectomy	28 hours	Lysis
38	24	Evanescent	Herniorrhaphy	28 hours	Crisis
39	46	Evanescent	Herniorrhaphy	36 hours	Lysis
40	12	Evanescent	Appendectomy	18 hours	Crisis
41	18	Evanescent	Appendectomy	12 hours	Crisis
42	43	Evanescent	Cholecystectomy	18 hours	Lysis
43	22	Mild	Herniorrhaphy	20 hours	Lysis
44	32	Fulminant	Appendectomy	28 hours	Crisis
45	65	Mild	Cholecystectomy	18 hours	Lysis
46	18	Moderate	Herniorrhaphy	14 hours	Lysis
47	40	Fulminant	Appendectomy	18 hours	Crisis
48	35	Moderate	Appendectomy; suspension	14 hours	Lysis (48 hours)
49	21	Moderate	Appendectomy	16 hours	Lysis
50	47	Fulminant	Cholecystectomy	16 hours	Lysis

* N_2O—Ether is understood to mean a gas induction, which was followed by the use of ether vapor; Ben Morgan apparatus and the new type of McKesson machine were used.

† In this column, ♂ indicates male; ♀, female.

‡ This case was a true lobar pneumonia from the onset; no operative procedure previously.

TABLE 1.—*Summary of Fifty Case Histories—Continued*

Case	Location	Deaths	Anesthetic	Sex†	Disappearance Time	
					Clinical Signs	Physical Signs
1	Right base	No	N_2O—Ether*	♂	36 hours	60 hours
2	Right base	No	N_2O—Ether	♂	60 hours	5 days
3	Right base	No	N_2O—Ether	♀	36 hours	60 hours
4	Left base	Yes	Drop ether	♀	Expired (peritonitis and myocarditis)	
5	Right upper lobe	Yes	Local procaine, 1%, field block	♀	Expired (Bronchopneumonia and sepsis)	
6	Right base	No	N_2O—Ether	♀	3 days	5 days
7	Right middle and base	No	N_2O—Ether	♂	6 days	13 days
8‡	Right middle and base	No	Postpneumonic	♂	Still showed pathologic condition on leaving hospital	
9	Right upper lobe	No	N_2O—Ether	♂	3 days	7 days
10	Right middle and base	No	N_2O—Ether	♂	2 days	4 days
11	Left base	No	Drop ether	♀	36 hours	48 hours, except the heart still to left
12	Left upper lobe	No	N_2O—Ether	♂	48 hours	60 hours
13	Left base	No	N_2O—Ether	♂	36 hours (1st) 72 hours (2d)	8 days in all
14	Right upper and base	No	Drop ether	♂	50 hours	72 hours
15	Right base	No	N_2O—Ether	♂	5 days	9 days
16	Right base	No	N_2O—Ether	♂	3 days	10 days
17	Left base	Yes	N_2O—Ether	♀	Expired (general peritonitis)	
18	Right middle and base	No	Local procaine, 1%, block	♂	5 days	7 days
19	Right base	No	N_2O—Ether	♂	5 days	7 days
20§	Right base	Yes	Spinal; 10 cc. 1% procaine	♂	Cleared up; then developed pneumonia in left lung and general peritonitis	
21	Right middle and base	No	N_2O—Ether	♂	3 days	5 days
22	Right upper and base	No	N_2O—Ether	♀	4 days	7 days
23#	Right base	No	N_2O—Ether	♀	5 days	8 days
24	Left base	No	N_2O—Ether	♀	3 days	5 days
25	Right middle and base	No	N_2O—Ether	♂	48 hours	72 hours
26	Left base	No	Drop ether	♂	36 hours	60 hours
27	Right base	No	N_2O—Ether	♀	36 hours	60 hours
28	Left base	No	N_2O—Ether	♂	24 hours	48 hours
29	Right base	No	N_2O—Ether	♂	36 hours	60 hours
30	Left base	No	N_2O—Ether	♂	2 days	5 days
31	Right base	No	Local block procaine 1%	♀	2 days	4 days
32	Left base	No	N_2O—Ether	♀	2 days	5 days
33	Right base	No	N_2O—Ether	♀	2 days	4 days
34	Right base	No	N_2O—Ether	♀	4 days	8 days
35	Right base	No	N_2O—Ether	♀	4 days	8 days
36	Right base	No	N_2O—Ether	♂	36 hours	48 hours
37	Right base	No	N_2O—Ether	♂	3 days	6 days
38	Left upper lobe	No	N_2O—Ether	♂	18 hours	36 hours
39	Right base	No	N_2O—Ether	♂	18 hours	28 hours
40	Right base	No	N_2O—Ether	♂	18 hours	28 hours
41	Right base	No	N_2O—Ether	♀	36 hours	48 hours
42	Right base	No	N_2O—Ether	♂	24 hours	36 hours
43	Right base	No	N_2O—Ether	♂	36 hours	48 hours
44	Right base	No	N_2O—Ether	♂	40 hours	72 hours
45	Left base	No	N_2O—Ether	♀	48 hours	4 days
46	Bilateral	No	Spinal; 1% 10 cc. procaine	♂	72 hours	5 days
47	Right middle and base	No	Procaine and gas	♂	48 hours	4 days
48	Left upper	No	N_2O—Ether	♀	48 hours	4 days
49	Right base	No	N_2O—Ether	♀	50 hours	4 days
50	Right middle and base	No	N_2O—Ether	♀	72 hours	5 days

§ This patient was operated on under spinal anesthesia; developed a collapse which cleared up entirely, but subsequently developed a bronchopneumonia on the left, in conjunction with a peritonitis.

This patient developed a purulent bronchitis after the collapse had entirely disappeared.

the 9th, when he suddenly became cyanotic, dyspneic and began coughing, complaining of pain in the chest at the same time. Physical examination revealed some dulness in the right base with distant breath sound. During the night, he spit up considerable thick, tenacious, mucopurulent material, and in the morning roentgen-ray evidence was negative but still showed a few râles. Two days later the chest was found to be entirely clear.

CASE 48.—A white boy, aged 12, was admitted to the hospital, Feb. 14, 1927. The diagnosis was subacute appendicitis. Operation was performed the following day under drop ether; the anesthesia lasted thirty minutes. At the time of the

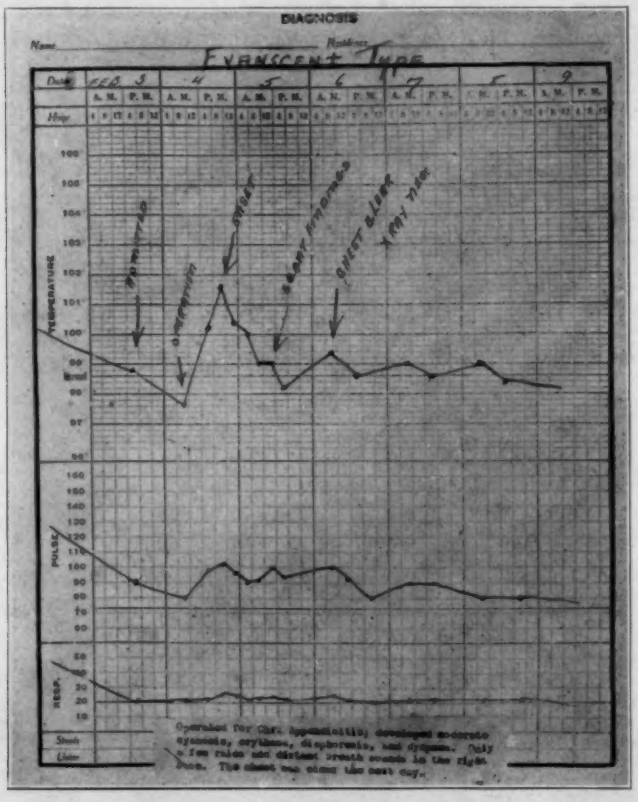

Fig. 18 (case 49).—Summary of course of atelectasis.

operation, the temperature was 98.4; pulse, 86, and respirations, 22. The patient apparently was doing nicely until the early morning of February 16, when he became somewhat cyanotic and dyspneic and had facial erythema. The temperature was 100.6; pulse, 104, and respirations, 30. On examination that morning, the breath sounds were found suppressed on the right and there was some limitation of motion in the right side of the chest.

He expectorated some plugs and a moderate amount of thick, tenacious material. The following day the chest was reported as clear. A roentgenogram on February 27 showed a normal chest.

CASE 49.—A white girl, aged 18, was admitted to the hospital, Feb. 7, 1927. She was operated on the next day under drop ether, open method. The anesthesia lasted for thirty-five minutes. The temperature was 98.8; pulse, 86, and respirations, 18, the day of the operation. The night of the following day and early in the morning, the temperature was 101.8; pulse, 102, and respirations, 26. A profuse diaphoresis was present, and the patient complained of tightness in the chest. She coughed very little. The next morning, nothing but a few râles was present in the right base. She expectorated several times during the day, and was somewhat erythematous. Nothing else of importance could be found.

The patient went on to an uneventful and smooth recovery.

Cholecystectomy was performed, January 27, under spinal anesthesia; difficulty was encountered, and closure was effected under gas (N_2O) anesthesia,

CASE 50.—A white man, aged 43, was admitted to the hospital, Jan. 25, 1927. which lasted fifteen minutes. About sixteen hours later there was a sudden rise in the temperature to 100.8; the pulse rate was 114; respirations, 26, and there was some feeling of tightness in the throat and chest. Considerable diaphoresis and some erythema and cyanosis were present; the next day, a few râles were heard in the right side of the chest; there was suppression of the breath sounds in the lower part of the right side of the chest.

The patients coughed up considerable thick, mucopurulent material and felt relieved. The next day about the same symptom complex was present, but the condition cleared up in about twenty-four hours. The temperature was 102.4; pulse, 120, and respirations, 28, the second time at 11 p. m. The chest was found to be clear the next day.

Roentgen-ray examination showed only suggestive displacement of the heart and mediastinal structures to the right.

GLOSSOPHARYNGEAL NEURALGIA (TIC DOULOUREUX)

ITS DIAGNOSIS AND TREATMENT *

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The pain of trigeminal neuralgia was for many years considered so distinctive in type and severity as to deserve the restrictive designation, "tic douloureux." Why this particular nerve should alone be subject to this paroxysmal, ticlike type of pain and all the other cranial and spinal sensory nerves be seemingly immune, has never had a rational explanation.

As a matter of fact, pain of the same type does occur in the distribution of the glossopharyngeal nerve, so that the term "tic douloureux" is no longer synonymous with trigeminal neuralgia, but must now include glossopharyngeal neuralgia. Whether similar pains of the facial, vagus and possibly of other sensory nerves will eventually be found to be of similar kind, remains to be seen.

Weisenburg [1] was apparently the first to direct attention to the resemblance of an unusual pain of the ninth nerve to the so-called idiopathic pain of tic douloureux of the trigeminal nerve. In his patient, however, the paroxysms of pain were caused by a tumor in the cerebellopontile angle, but in many ways the pain was analogous to that of tic douloureux of the trigeminal nerve. In fact, it was first mistaken for trigeminal neuralgia, but the pain was not influenced by a partial removal of the gasserian ganglion. The pain was always at the "root" of the tongue, and extended down the throat and to the ear. There was a burning feeling and dryness in the throat, and frequently the sensation of flies and roaches crawling. At times the paroxysms were excruciating, and usually were brought on by eating. During the last year of the patient's life, contact of food with a sharply defined trigger zone at the base of the tongue caused terrific paroxysms of pain. Actual chewing movements did not induce this pain.

Ten years after Weisenburg's report, three cases of "algie vélopharyngée essentielle" were described by Sicard and Robineau.[2] In each case the pain was paroxysmal, was induced by swallowing, chewing, speaking and often spontaneously, and was referred to one side of the pharynx, soft palate and tonsil. Doubtless because the pain was of

* From the Johns Hopkins Hospital and University

1. Weisenburg, T. H.: Cerebello-Pontile Tumor Diagnosed for Six Years as Tic Douloureux: The Symptoms of Irritation of the Ninth and Twelfth Cranial Nerves, J. A. M. A. **54**:1600 (May 14) 1910.

2. Sicard, R., and Robineau: Communications et presentations: I. Algie vélopharyngée essentielle. Traitement chirurgical, Rev. neurol. **36**:256, 1920.

long duration and not accompanied by other manifestations of tumor, the condition was considered to be "essential" or idiopathic. It is hardly probable that they recognized the pain as an involvement solely of the glossopharyngeal domain, for their treatment, as practiced and recommended, consisted in sectioning in the neck the branches of the glossopharyngeal nerve, the pharyngeal branches of the vagus and the superior cervical ganglion. Since the appearance of these publications, a number of cases have accumulated rapidly.

Doyle [3] reported four and Adson [4] three other cases from the Mayo Clinic. Harris,[5] in his splendid book, "Neuritis and Neuralgia," adds six cases from his own service in London. As this paper was written another case was appended by Goodyear.[6]

Although all of these have been reported as cases of glossopharyngeal neuralgia, the doubt lurks that the pain is confined exclusively to the distribution of the glossopharyngeal nerve. For this reason, the suggested treatment of Sicard and Robineau was followed in the cases of Doyle and Adson, i. e., the branches of both the vagus and glossopharyngeal nerves were divided. They seem to have been misled by an uncertainty of the sensory nerve supply to the pharynx, believing branches of the sphenopalatine ganglion and vagus nerve to be the source of its supply. Harris is "uncertain whether the vagus has any share in the production of this (glossopharyngeal) neuralgia, and whether section of the pharyngeal branch of the vagus is also necessary." It appears probable that the unorthodox and faulty conclusions by Vernet [7] (referred to by Sicard and Robineau and by Doyle) on the nerve supply to the pharynx by the glossopharyngeal and vagus nerves is largely responsible for this confusion. The sensory distribution of the ninth nerve will be considered later.

In addition to the foregoing series of cases assembled from the literature, two more are reported. Curiously, these cases appeared six weeks apart. Both were recognized as glossopharyngeal neuralgia by the characteristic history of the attacks before the patients were actually seen. These cases are presented not only to emphasize the clinical picture of this remarkable and not uncommon condition, but also to present a simple form of treatment, practically devoid of danger, and productive of permanent cure without disability.

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3. Doyle, J. B.: A Study of Four Cases of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. **9**:34 (Jan.) 1923.

4. Adson, A. W.: The Surgical Treatment of Glossopharyngeal Neuralgia, Arch. Neurol. & Psychiat. **12**:487 (Nov.) 1924.

5. Harris, W.: Neuritis and Neuralgia, New York, Oxford University Press, 1926.

6. Goodyear, H. M.: Tic Douloureux of the Glossopharyngeal Nerve, Arch Otolaryng. **5**:341 (April) 1927.

7. Vernet, M.: Syndrome du trou déchiré postérieur (Paralysie des nerfs glossopharyngien, pneumogastrique spinal), Rev. Neurol. **34**:117, 1918.

CASE 1.—*History.*—A strong, healthy man, aged 45, but looking much older, was first seen in the neurologic dispensary by Dr. Frank Ford, who at once recognized the pain to be that of glossopharyngeal neuralgia. Seldom have we seen a man in greater agony, even in the most severe cases of trigeminal neuralgia. He was then having paroxysms of terrific pain in rapid succession, some induced by swallowing or talking and some even occurring spontaneously. Afraid to swallow or to talk, he sat in terror, with his head hanging forward and directed toward the right in order to allow the saliva to drool from his mouth and away from the affected side. He dared to eat only after his tongue had been cocainized over its base, and particularly, as he so strongly emphasized, over one spot about the size of a dime, near the junction of the tongue with the left tonsil. This "trigger zone," so real to him, did not show anything on examination. For three and one half years he had endured this pain at varying intervals. It had begun in September, 1923, with a severe knifelike thrust at the base of the tongue immediately after taking a drink of cold water; it lasted a few seconds, but for three weeks it occurred several times a day and ended as abruptly as it began. During the attacks, the left ear drum felt as though it were being pushed out and "were ready to burst." In one severe attack just before his operation, the patient felt as if all the teeth in the lower jaw on the affected side were "jumping out of their sockets." For more than two years he was free from pain when asleep; but during the six months before admission to the hospital, he had been frequently wakened from a sound sleep by the knifelike pains, particularly when lying on the left side. The attacks were due, he thought, to the accumulation of saliva on the trigger zone. His tonsils had been removed in September, 1926, and again in November, 1926, in the vain hope of effecting a cure. During the latter operation, the throat was extensively burned with a cautery. The pain was decidedly worse afterward, and from then until his admission to the Johns Hopkins Hospital in April, 1927, the pains became more and more frequent and severe. He said it seemed to him that a red hot poker was being jabbed through the tongue. At first the attacks were of only two or three seconds' duration, but during the past six months each had lasted about ten seconds. He had the impression that the attacks were more frequent when he talked a great deal, but in his occupation as a missionary there was little opportunity to get complete rest for any length of time. He had obtained relief for a few days by local application of cocaine.

Results of all examinations, including tests for taste and sensation in the domain of the ninth nerve, were entirely negative. The Wassermann reaction was negative.

Operation.—On April 6, 1927, I performed an intracranial section of the ninth nerve, in the posterior cranial fossa (fig. 1). The patient made an uneventful recovery, with immediate and subsequent complete relief from pain of any kind. There was no disturbance of swallowing afterward. He was unable to detect any symptoms due to the loss of function of the ninth nerve. Objectively, however, there was total loss of sensation and of taste over the back of the tongue and pharynx (fig. 6, 7 and 8). No motor impairment of any kind could be detected by examination.

CASE 2.—A man, aged 56, well developed and strong, but pale and sallow from lack of nourishment, was referred by my associate, Dr. S. J. Crowe, on May 10, 1927. Fifteen years before, while talking with a friend, the patient had been suddenly seized with an excruciating pain in a spot at the back of the tongue and near the right tonsil. It lasted only a few seconds, but during the following years similar pains in the same location struck him suddenly without warning;

after they had disappeared, he was entirely free of pain. It seemed as though a red hot iron were being thrust through the tongue at this spot. At first he was unable to discover any inciting cause for the attacks. The free intervals were as long as a year and a half, but the pain always recurred in the same spot. Since November, 1926, the attacks had become more frequent and severe and of longer duration, often lasting several seconds. During this time, the attacks have been induced consistently by drinking, eating and talking. For two weeks before I saw the patient, he had been almost without food or drink and had scarcely dared to speak. Sneezing and coughing also brought on the paroxysms. For the two days before he consulted me, one attack had followed almost immediately

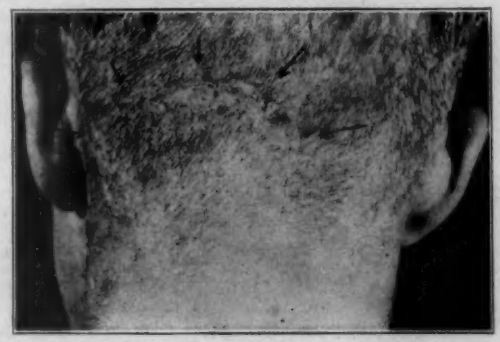

Fig. 1.—Posterior view of patient's head to show the operative scar, indicated by the arrows. The photograph was taken on discharge from the hospital ten days after the operation.

Fig. 2.—Scar of second patient when ready for discharge two weeks after operation. The arrows indicate the position of the scar.

on the other, giving the patient almost no interval of relief longer than a few minutes, despite hypodermic injections of morphine. During my brief examination the patient had three paroxysms, each lasting about five seconds. Immediately after each attack, he could swallow with impunity for a few seconds, but this period of immunity was soon over. He could direct the examiner to the spot on the base of the tongue which seemed to him so well defined, but nothing was objectively evident. Results of all examinations, including tests for taste and sensation, in the distribution of the ninth nerve were negative.

Operation.—On May 11, 1927, under local procaine hydrochloride anesthesia, the glossopharyngeal nerve was divided intracranially in the lateral cistern (fig. 2). Postoperative convalescence was uneventful. All pain immediately dis-

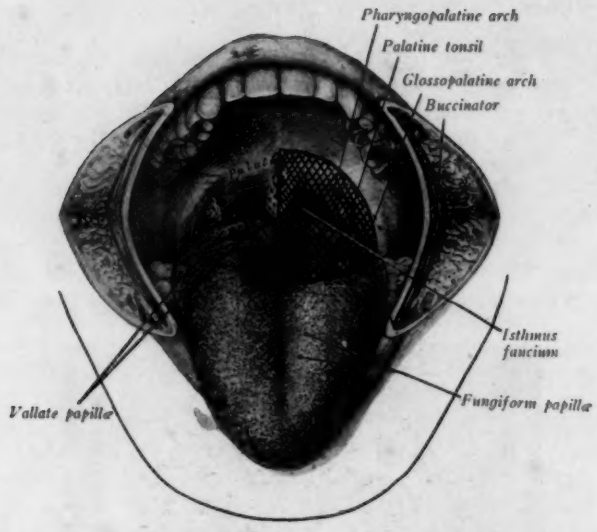

Fig. 3.—Area of anesthesia in the mouth following unilateral division of the glossopharyngeal nerve. (Photograph from Gray's "Anatomy.")

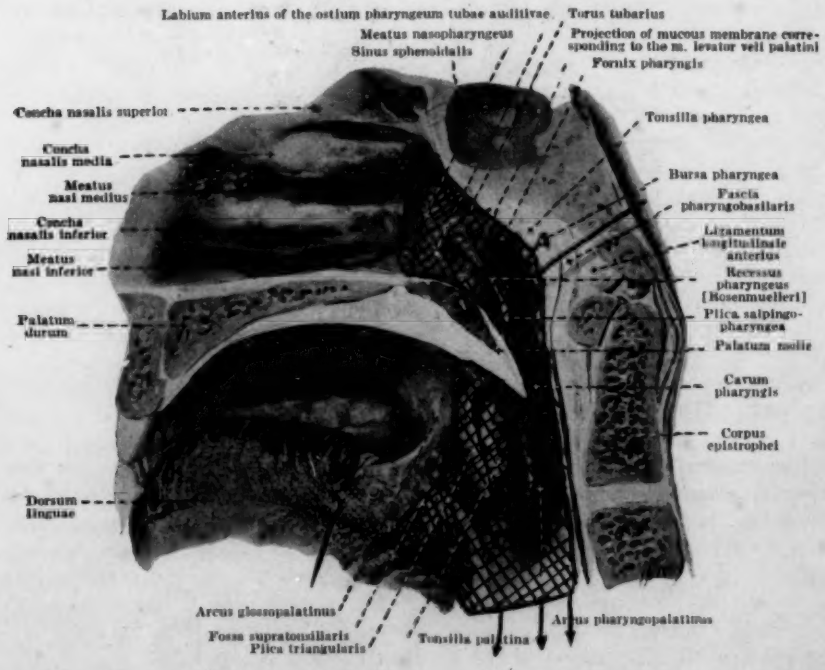

Fig. 4.—Cross-hatched area indicates area of anesthesia in the pharynx and nasopharynx, lateral view. Sensation is uncertain in the area shown in dots at the vault of the pharynx. (Photograph from Spalteholtz.)

appeared and had remained absent up to June 1, 1927. The patient was unable to detect any abnormal sensations incident to the loss of sensation. There were no signs or symptoms of injury to the vagus nerve. The accompanying illustrations, figures 3, 4 and 5, show the sensory changes due to the loss of the ninth nerve. Motor loss could not be observed. The patient was a public speaker, and, despite his terrible pain, was most reluctant to have anything done unless assured that his voice would be unimpaired; there was no change.

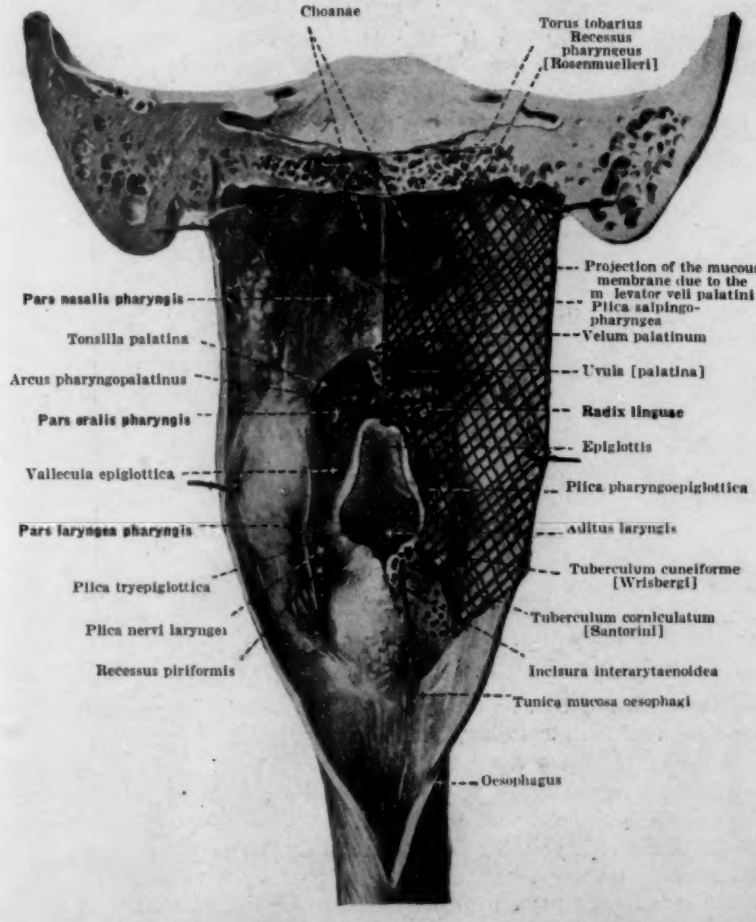

Fig. 5.—Showing area of anesthesia in the pharynx (looking from behind) after division of the glossopharyngeal nerve. Sensation is uncertain in the area over the esophagus, shown in dots. (Photograph from Spalteholtz.)

THE OPERATION

The glossopharyngeal nerve is divided intracranially, a unilateral cerebellar approach being used. It is the same exposure that I have used for the past three years as the method of choice in dividing the sensory root of the trigeminal nerve. The glossopharyngeal nerve is, however, much easier of access. It is one of the simplest of intracranial operations, and can readily be performed with

either local or general anesthesia. An incision is made which begins at a point about 2 centimeters below the inion, gently curves upward, reaching slightly above the line of attachment of the trapezius muscle to the occipital bone, and then turns more abruptly downward along the mastoid bone until within from 1.5 to 2 centimeters of its tip (fig. 6). The trapezius muscle is divided just below its attachment and retracted downward and outward. The bone is removed over much of the corresponding side, at least far enough to the midline to afford access to the cisterna magna, which is punctured to release fluid and to provide additional room. The bone is rongeured laterally to the mastoid, but not far enough to enter the mastoid cells. The dura is opened in stellate fashion. Gentle elevation of the cerebellar lobe then allows ample exposure of the cerebellopontile angle. The cisterna lateralis is quickly brought into view, and, when pricked, the lining arachnoid membrane collapses (fig. 7). The fifth, eighth, ninth, tenth and

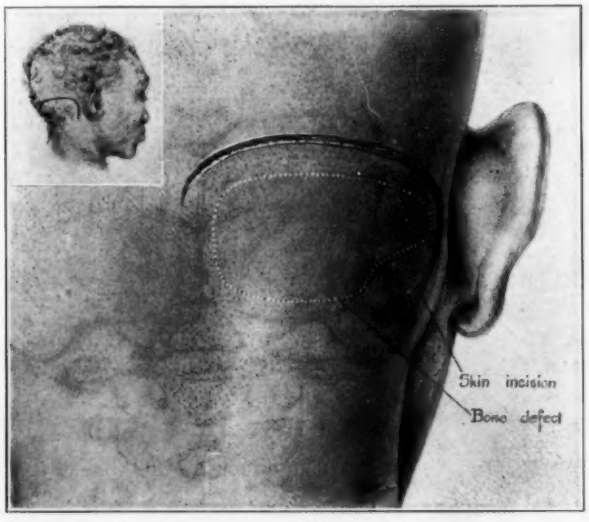

Fig. 6.—Outline of incision in skin and defect in bone in operation for intracranial section of the glossopharyngeal nerve.

eleventh cranial nerves then appear. The auditory nerve is probably the best landmark. The glossopharyngeal nerve is, of course, next in succession in a caudal direction and is probably from 0.75 to 1 centimeter caudal to the auditory nerve. A relatively tiny nerve, about the size of the lead in a pencil, always single and free from contact with the vagus, which lies immediately caudal—the glossopharyngeal nerve—can be readily lifted with a blunt hook without touching the vagus or any other nerve. A knife or a pair of scissors cuts the suspended nerve in an instant (fig. 8). The patient whose nerve was sectioned under procaine hydrochloride anesthesia (applied only extracranially) did not experience any sensation when the glossopharyngeal nerve was divided. The operation should be devoid of danger. There is no reason for even touching the vagus, auditory or facial nerves. There can, of course, be no question about the permanency of a cure, for precisely the same problem has long since been solved in trigeminal neuralgia. After section of the sensory root of any nerve central to its ganglion, regeneration is impossible.

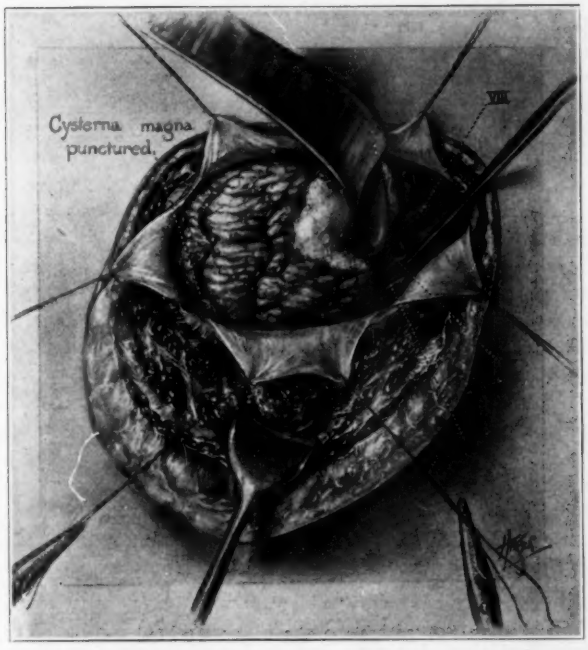

Fig. 7.—Elevation of the cerebellum, exposing the cerebellopontile angle after puncture of the cisterna magna and the cisterna lateralis. The ninth nerve is shown drawn forward with the knife hook. The eighth and tenth nerves are always plainly in view. The ninth nerve is separated from the vagus by 2 or 3 mm., thereby preventing any injury to the vagus during its manipulation and division.

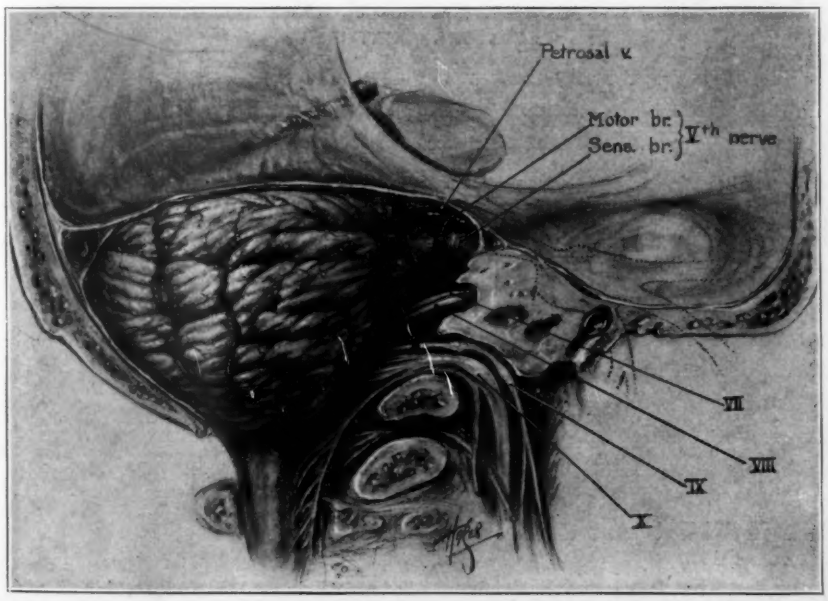

Fig. 8.—Point of division of the glossopharyngeal nerve intracranially. By the same exposure, the fifth nerve may be divided in trigeminal neuralgia without injury to the motor branch, which is a few millimeters distant.

The operations heretofore employed in glossopharyngeal neuralgia have been of the peripheral type, either section or avulsion of the nerve. Besides the local difficulties of isolating and dividing the ninth nerve high up in the neck without injuring the sympathetic trunk and particularly the vagus (a difficult task), the objection remains that regeneration of the nerve is likely to follow its division peripherally. As noted before, Sicard and Robineau introduced this manner of division of the nerve for glossopharyngeal neuralgia, and in all cases the sympathetic and pharyngeal fibers of the vagus were intentionally included. Adson avulsed the glossopharyngeal nerve in the hope that at least the petrous ganglion would be included, but the vagus, being closely associated, was injured. He regretted that he had not tried intracranial section of the nerve and hoped to do so in his next case. In a patient afflicted with an extensive carcinoma of the tongue and pharynx, Fay,[8] in 1926, sectioned the ninth nerve, together with three upper cervical nerves, in an extensive occipital and cervical exposure. The vagus nerve was injured, and, doubtless because of inadequate exposure, the pulse rate immediately dropped from 125 to 80. He also planned a unilateral cerebellar approach if a patient with glossopharyngeal tic douloureux should come for operation. Harris, in 1926, attempted to inject the glassopharyngeal nerve (in the neck) with alcohol, but failed to reach the nerve. Injections of alcohol could hardly be successful. Injury to the contiguous jugular vein, the internal carotid artery, the sympathetic, spinal accessory and vagus nerves would seem more probable than a successful injection into the glassopharyngeal nerve.

In passing, it is well to note the importance of section of the glossopharyngeal nerve in inoperable carcinomas of the tonsil and nasopharynx. Should the domain of the fifth nerve also be invaded by the growth, the sensory root of the trigeminal nerve can easily be divided at the same time and with little added time. As mentioned before, this approach has been my method of choice for section of the sensory root in tic douloureux for the past few years.

CLINICAL FEATURES

The close similarity between glossopharyngeal neuralgia and tic douloureux of trigeminal origin has been commented on by Sicard and Robineau, Doyle, Adson and Harris. Surely both varieties of this fearful pain equally deserve the appellation, tic douloureux. The characteristic sudden, ticlike paroxysms of excruciating pain and the induction of pain by external stimuli are common to each.

In the twenty cases of glossopharyngeal neuralgia assembled from the literature and epitomized in the accompanying table, the clinical picture is strikingly uniform. The paroxysms of pain always strike some part of the glossopharyngeal sensory area—the tonsil, back of the tongue or the pharynx; the pain is always unilateral and induced by liquids or solids passing over the sensory endings of the ninth nerve in the mouth or pharynx. The pain is always excruciating; it is variously described as lancinating, knifelike or like stabs of a red-hot iron. Each individual pain usually lasts only a few seconds, rarely over a

8. Fay, T.: Intracranial Division of Glossopharyngeal Nerve Combined with Cervical Rhizotomy for Pain in Inoperable Carcinoma of the Throat, Ann. Surg. **84**:456, 1926.

minute; the duration of the paroxysm, is, therefore, usually less than that of trigeminal neuralgia, but it would seem more severe, if possible. There is a strong tendency for the paroxysms to occur in a series over a period of time varying from a few days to a few weeks; in nearly every case intermissions of several months have occurred. The longest period of relief was three and three-fourths years in one of Adson's cases. But the identical pain always returns in precisely the same location. In a few cases there has been some tendency for the pain to shift from an initial point more remote and eventually to reach the central area of the glossopharyngeal sensory domain; for example, in two of Doyle's cases the original pain was in the ear, and in one of Adson's cases it was in the lobule of the ear and the angle of the mandible, but the pains were brought on by swallowing or other stimuli of the tongue and pharynx. In nearly all cases pain radiates to the ear, i. e., the meatus, the concha, the lobule or in front of or behind the ear, and not infrequently it begins there either before the more central pain or almost simultaneously with it. Rarely does the pain radiate elsewhere than to the ear or its environs and to the affected side of the throat. It seems probable that Jacobson's nerve is responsible for this aspect of the pain, but as there is no external sensory supply by the glossopharyngeal nerve, pain to the mandible and outer ear must be looked on as referred. At times, as in two of Doyle's cases, in one of Adson's and in one of Harris', the ear was actually tender to touch. In the latter two instances, touching or washing the ear would precipitate the characteristic attacks. Although the method par excellence of starting attacks of glossopharyngeal neuralgia is to drink hot or cold liquids—cold is more effective than hot—the attacks are also induced in many other ways. Other recognized causes are: swallowing (solid foods seem to be less effective than liquids), talking, yawning, coughing, shouting, sneezing, touching the ear, suddenly turning the head, and in one instance touching the angle of the mandible; often the attacks seem to occur spontaneously. A trigger zone was present in both of the cases reported (the base of the tongue, near the tonsil), in one of Adson's cases (pharyngeal wall) and in Goodyear's case (tonsillar pillar). Harris remarked on the absence of a "trigger zone" in cases of glossopharyngeal neuralgia, and considered its absence to be almost the only essential difference between glossopharyngeal neuralgia and trigeminal neuralgia; the four cases in which there were trigger zones have appeared since the publication of his book.

Like trigeminal neuralgia, glossopharyngeal neuralgia usually appears in the later half of life, and usually after the age of 50. Whether significant or not, one observation stands out strikingly in this early series; there are only three cases (10 per cent) in women.

I have placed in the same group the cases in which the glossopharyngeal neuralgia was caused by a tumor and those in which it has been considered essential or idiopathic. As a matter of fact, the only reason for having assumed that the condition is idiopathic was the analogy of the features of the pain with those in trigeminal neuralgia. Necropsy was performed only in Weisenburg's case; and an intracranial exposure of the ninth nerve has been made only in my two cases. From the latter cases alone, positive objective proof is furnished of the essential nature of glossopharyngeal neuralgia. As a matter of fact, the proportion of tumors that have been demonstrated is high, compared with their incidence in trigeminal neuralgia. Three of the series of patients are known to have had tumors: Weisenburg's patient with cerebellopontile tumor and two of Harris' patients who had carcinoma of the tonsil. It seems probable that tumors were present in two more instances. One of Harris' patients lost 22 pounds (10 Kg.) in weight in two months, indicating a serious wasting underlying cause. Another victim had convulsions induced by the pain; in fact, a cerebral exploration had been made for a tumor of the brain, though with negative results. This patient died a year later, presumably from the intracranial lesion (tumor?). There is, therefore, a known incidence of 15 per cent of tumors in this group and a probable incidence of at least 25 per cent. Weisenburg's case, taken in conjunction with the well known slow, insidious growth of tumors in the cerebellopontile angle, as well as the frequently obscure malignant lesions of the tonsil and nasopharynx, should make one always alert to the possibility that an underlying tumor may be the cause instead of an idiopathic neuralgia. Moreover, a careful survey of the histories in the cases here assembled seems to indicate no discernible difference in the character or location of the pain or its provoking influences, whether there is an underlying tumor or not. One additional great advantage of the intracranial operation for glossopharyngeal neuralgia is that automatically any intracranial lesion is discovered and may be eliminated. If a tumor is causing the condition, no treatment is, of course, rational which does not deal with the removal of the tumor if that is possible.

The diagnosis of essential glossopharyngeal neuralgia is made solely from the history and from observation of the attacks. In many of the cases from the literature a diagnosis of trigeminal neuralgia was made, but this was before the glossopharyngeal neuralgia was recognized. The pain of glossopharyngeal neuralgia was so characteristically located in the region of the ninth nerve that in none of the cases collected here should there be any doubt of the diagnosis. One might possibly think of trigeminal neuralgia when the ear is tender and when touching the ear incites attacks, but even then the pain is mainly induced by stimuli in the back of the mouth, and the pain is always in this region.

Neuralgia of the seventh nerve (geniculate ganglion) has been described by Hunt as accompanied by pains largely localized in the ear.

Geniculate herpes is a well recognized condition, but geniculate neuralgia is none too securely established; at least it remains to be proved that a ticlike neuralgia of the seventh nerve occurs without more spasms. A tic of the facial nerve, described by Cushing as "tic conouloif," is characterized by spasms of the facial muscles, and cannot be confused with glossopharyngeal neuralgia.

GLOSSOPHARYNGEAL NERVE SUPPLY

Isolated loss of function of the glossopharyngeal nerve apparently has not been known. Pope,[9] in describing a case of thrombosis of the vertebral artery pressing on the glossopharyngeal nerve, quotes Gowers as saying: "There is no recorded case in which the roots of the glossopharyngeal nerve alone have been diseased." Cassirer,[10] writing in Oppenheim's Lehrbuch makes a similar statement. Cases of tumor or traumatic injuries to the nerves at the base of the skull have paralyzed the glossopharyngeal nerve, but this paralysis has never been purely and solely of the glossopharyngeal nerve. Indeed, Pope's case seems more probably an aneurysm and not a thrombosis, for there was a fusiform mass. In addition to the incomplete injury to the glossopharyngeal nerve, there was partial facial paralysis and some disturbance of the trigeminus, both subjective and objective; moreover, it is difficult to understand how the vagus, which is in such close proximity, could have been spared. It is almost inconceivable that any neoplasm could be so small and so isolated as to cause a pure glossopharyngeal paralysis, and it is equally incredible that any effect of trauma could induce an isolated paralysis of this nerve. Vernet's conclusions on glossopharyngeal function, as noted, which have received much more consideration than they deserve, have been derived from observations of tumors, inflammations and fractures affecting all of these structures in the jugular foramen. From such a source it is impossible to separate disturbances of the ninth and tenth nerves, which are so intimately related over such a long distance. The only possible way to cause pure glossopharyngeal destruction and, therefore, to test the function of this nerve is by intracranial section at operation. Section of the nerve in the neck has always been accompanied by injuries of the vagus and usually of the sympathetic nerve. The entire nerve can never be sectioned or avulsed in the neck, for branches—notably Jacobson's nerve—cannot be reached. The two

9. Pope, F. M.: Thrombosis of Vertebral Artery Pressing on Glossopharyngeal Nerve; Unilateral Loss of Taste at Back of Tongue, Brit. M. J. **2**:1148, 1889.

10. Cassirer: Die Lähmung des N. Glossopharyngeus, in Oppenheim, Lehrbuch der Nervenkrankheiten, 1923, vol. 1, p. 775.

Table of Cases of Glossopharyngeal Neuralgia Assembled from the Literature

Author and Year	Age and Sex	Duration of Pain	Nature of Pain	Location of Pain	Radiation of Pain	What Induces Pain	Duration of Paroxysm	Pain in Ear	Tenderness of Ear	Trigger Zone	Pain Unilateral	Pain Has Intermission	Cause of Pain	Treatment
Weisenburg 1910	35 ♂	.....	Sharp shooting; dry burning feeling in throat	Root of tongue	Throat and ear	Eating, drinking	....	+	....	+	+	..	Tumor of cerebellopontile angle	Gasserian ganglion
Sicard and Robineau 1920	Young soldier ♂	Several years	Severe paroxysm	Pharyngeal wall	.....	Swallowing, talking, chewing	....	..	....	..	+	+	None	Peripheral section of ninth, pharyngeal branches of tenth, and sympathetic nerves
Sicard and Robineau 1920	Young soldier ♂	Several years	Severe paroxysm	Pharyngeal wall	.....	Swallowing, talking, chewing	....	..	....	..	+	+	None	Peripheral section of ninth, pharyngeal branches of tenth, and sympathetic nerves
Sicard and Robineau 1920	51 ♀	.....	Severe paroxysm	Pharyngeal wall	.....	Swallowing, talking, chewing	....	..	....	..	+	+	None	Peripheral section of ninth, pharyngeal branches of tenth, and sympathetic nerves
Doyle 1923	63 ♂	5 yr.	Severe paroxysm	First in ear, later in tonsillar region	Ear	Eating, drinking, talking, yawning	....	+	+ of auricle	..	+	+	None	First gasserian, later peripheral section of ninth and tenth nerves
Doyle 1923	52 ♂	3 wk.	Severe paroxysm	First in ear, later in throat	Ear	Talking, swallowing, spontaneous	20 to 40 seconds	+	+ over tragus after paroxysms	..	+	..	None	Injection in third branch of fifth and auricular temporal nerves
Doyle 1923	57 ♀	1 yr.	Paroxysms; feeling of choking	Side of pharynx	Angle of mandible	Drinking	1 to 2 seconds	..	..	..	+	+	None	None
Doyle 1923	61 ♂	9 yr.	Excruciating paroxysms	Side of pharynx	External auditory meatus	Movements of head, talking, drinking, swallowing, fright	2 to 50 seconds	+	..	None	+	+ pains in series	None	None
Adson 1924	61 ♂	9 yr.	Severe paroxysms	Lobule ear and pharynx	Ear and angle of mandible	Movements of head, swallowing, talking, spontaneous	....	+ lobule	+ Excessive, auricle	None	+	+ longest 3¾ years	None	Attempted peripheral avulsion of ninth nerve, but nerve later found intact; vagus injured
Adson 1924	52 ♂	4 yr.	Severe stabbing pains	Tonsil and ear	Ear	Drinking, washing ear	....	+	+	+ on pharyngeal wall	+	+	None	Avulsion of ninth nerve; vagus injured

Adson 1924	38 ♂	17 yr.	Knifelike spasms	Pharyngeal muscles	Ear	Drinking, eating, talking, pressure on maxilla	Few seconds to few minutes	+	..	None	+	+ longest 6 mos.	None	Avulsion of ninth and part of tenth nerves
Harris 1923	87 ♀	12 yr.	Severe paroxysms	Left side of throat	Ear	Swallowing	....	+	Concha intensely tender to touch	None	+	+	None	Injection in mandibular branch of trigeminal nerve
Harris 1926	53 ♂	6 mo.	Severe paroxysms	Root of tongue.	Ear and back of jaw	Swallowing, eating, yawning	....	+	..	None	+	+	None	Attempted injection in ninth nerve not successful
Harris 1926	50 ♂	5 yr.	Excruciating paroxysms	Throat	Ear, back of jaw and neck	Swallowing and touching ear	....	+	Touching ear started attacks	None	+	+ longest 1 year	None; but had convulsions and died 2 years later (brain tumor ?)	Injection in mandibular nerve
Harris 1926	65 ♂	6 yr.	Severe paroxysms	Tonsil	Ear	Yawning but not swallowing	20 seconds	+	..	None	+	+ longest 1 year	Carcinoma of tonsil	Local removal of carcinoma
Harris 1926	59 ♂	.....	Pain on swallowing	Tonsillar region	.....	Eating, swallowing, hot drinks especially	....	..	None	None	+	..	Carcinoma of tonsil	Local removal of carcinoma
Harris 1923	54 ♂	.....	.....	Side of throat	.....	Excitement, muscular exertion; not swallowing	....	0	None	None	+	+	Possibly carcinoma; great loss of weight	None
Goodyear 1927	74 ♂	.....	Excruciating spasms	Throat and tonsil	Anterior to ear	Talking, swallowing, not chewing	+	..	..	+ tonsillar pillar	+	+	None	None
Dandy 1927	45 ♂	3½ yr.	Excruciating paroxysms	Back of tongue and tonsil	Ear drum feels as if pushed out	Swallowing, talking, spontaneous	Few seconds	+	None	+ base of tongue near tonsil (very striking)	+	+ longest 2 years	None	Intracranial section of ninth nerve
Dandy 1927	56 ♂	15 yr.	Excruciating spasms like thrust of hot iron	Base of tongue and tonsil	.....	Swallowing, talking, sneezing, coughing, spontaneous	2 to 5 seconds	..	None	+ base of tongue near tonsil (very striking)	+	+ longest 1½ years	None	Intracranial section of ninth nerve

cases here reported are presented as pure isolated lesions of the glossopharyngeal nerve; there were no other involvements of the cranial nerves. The after-effects of division of the glossopharyngeal nerve intracranially were almost the same in both cases.

Motor Changes.—Symptomatically, there was absolutely no subjective or objective motor disturbance in either instance. Swallowing was unaffected. The constrictors of the pharynx were unimpaired. The soft palate moved normally and equally on the two sides. Most textbooks of anatomy agree that the stylopharyngeus muscle is the only muscle supplied by the glossopharyngeal nerve. I do not know of any test that will demonstrate its loss of function; apparently its absence (if the nerve is affected) is not noticeable to the patient. These cases, I believe, effectually contradict the claims of Vernet that the important middle constrictor of the pharynx is supplied by this nerve; its nerve supply is from the vagus.

Alterations of Taste.—The nerve supply of taste will shortly be the subject of a more detailed study in conjunction with Dr. Dean Lewis. For this reason, only cursory mention will be made here of the part played by the ninth nerve in the supply of taste to the tongue as a whole.

In both of the cases reported there was complete loss of taste in the posterior third of the tongue. This confirms the usual conception of the nerve supply of taste for this part of the tongue. It opposes the rather unusual view long taught by Gowers and others that the trigeminal nerve supplies taste to the whole tongue. In one of the cases reported here, no change occurred in the perception of taste in the anterior two thirds of the tongue. In the other case there was a slight, but definite, alteration to the degree that on the normal side, sweet, sour, bitter and acid were all recognized more quickly (a few seconds) and registered with greater intensity than on the side with the cut glossopharyngeal nerve; but all tests for taste were accurately registered on the affected side (anterior two-thirds). The interpretation of these minor alterations of taste in one case and none in the other will be discussed in the forthcoming paper. At least, it is evident that the ninth nerve supplies taste for the posterior third of the tongue, but does not supply taste to the whole tongue, a view long held by Luciani and by his followers even at present.

Sensory Changes.—The results in both cases are best shown in the accompanying sketches. For the examinations of the sensory loss in the pharynx and nasopharynx and the base of the tongue I am indebted to Dr. S. J. Crowe. The only differences noted in the two cases were that in one patient sensation was perceived in the eustachian tube for a few millimeters below the pharyngeal orifice; in the other, there was no sensation for a distance of about 2.5 cm. In one patient the sensory deadline exactly bisected the uvula; in the other, almost the

entire uvula was anesthetic. In both patients, a cap of sensation remained at the vault of the nasopharynx, doubtless being supplied by the trigeminal nerve. The remainder of the pharynx, anterior, posterior and lateral, down to the epiglottis (including the posterior aspect), the vellecula and the pyriform sinus, was anesthetic. Anteriorly and posteriorly, the line of anesthesia ended sharply at the midline. The soft palate is supplied by the glossopharyngeal nerve only in a narrow rim on its oral surface and over a greater extent on its nasal surface. The tonsil and eustachian orifice were insensitive in both cases. The epiglottis was anesthetic on its posterior aspect; the line of demarcation of the area with the normal sensation of the anterior surface from that supplied by the vagus is a sharp line along the rim of the epiglottis. The precise line of demarcation between the area of normal and of lost sensation was difficult to determine in two places—above at the vault of the nasopharynx and below at the beginning of the esophagus. These two narrow zones are therefore shown in dots in contrast to the cross-hatch which denotes the absolute loss of sensation. The anterior wall of the sphenoid and the posterior end of both the middle and interior turbinates were sensitive. There was no area of anesthesia in the nasal cavity. It could not be determined whether there was any loss of sensation along the posterior border of the vomer.

Secretory and Sympathetic Nerves.—No disturbance of salivary secretion could be detected after the glossopharyngeal nerves had been sectioned.

I am aware that the small superficial petrosal nerve is a continuation of Jacobson's nerve through the tympanic plexus. It has received much attention as an important link in the chain of certain theories of taste; it has been supposed also to carry sensation to the nasal cavity, even to the pharynx. I have not been able to adduce evidence of either function.

SUMMARY AND CONCLUSIONS

1. Glossopharyngeal neuralgia is a type of tic douloureux exactly like trigeminal neuralgia. Its clinical picture is so characteristic and the history of the attacks so vivid that the diagnosis is easy and unmistakable.

2. Two cases are reported and appended to eighteen others which have been assembled from the literature. The clinical features of this condition are analyzed and tabulated.

3. The treatment of glossopharyngeal neuralgia is purely surgical. An operation by which the ninth nerve is sectioned intracranially was carried out in both cases. The superiority of this operation over section of the glossopharyngeal nerve in the neck is due to the fact that other nerves are not injured and the nerve is cut above the ganglion,

thereby precluding return of the malady. The operation is practically without danger to life and leaves no subjective or objective disturbance in its wake.

4. These isolated glossopharyngeal lesions afford an excellent opportunity to study the function—sensory, motor and gustatory—of this nerve. It is probable that these cases are the only instances of pure, unmixed and total loss of function of the ninth cranial nerve.

EXPERIMENTAL OBSERVATIONS ON THE GROWTH OF LONG BONES

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Our attention was attracted to the question of growth of bone by Haas' article in the ARCHIVES OF SURGERY,[1] in which he reviewed fully the large amount of experimental work which had previously been done. It is of interest to note that the first work of this type was done by Duhamel in 1739, and that John Hunter made some careful experiments on growth of bone ninety years ago. While his results—still preserved in the Hunterian Museum—do not show any longitudinal increase in the shaft of bone in pigs and fowls, he concluded nevertheless that some growth can occur. Ollier,[2] a young Frenchman from Lyons, coming to Paris in 1857, attracted by that genius of medical research, Claude Bernard, plunged into this fascinating problem in the laboratory of Chaveau. After a series of experiments with periosteum, he became completely convinced of its osteogenic properties. He also noted that irritation of the shaft of a bone could cause increase in the rate of growth of the epiphyses, and that it might even cause an overgrowth of as much as one-fifteenth of the total length of the bone. He repeated Duhamel's experiments to make certain in his own mind that longitudinal growth took place only at the epiphyseal lines.[3] He found slight separation of markers placed in ends of the shafts of young fowls, but not enough to change Duhamel's rule of nonelongation.

In spite of this experimental evidence, clinical observers have felt that, although most of the growth took place in the epiphyses, still a small amount could occur in the shaft. Truesdell,[4] Speed,[5] David [6] and

1. Haas, S. L.: Interstitial Growth in Growing Long Bones, Arch. Surg. **12:** 887 (April) 1926.

2. Keith, Arthur: Bone Growth and Bone Repair, Brit. J. Surg. **6:**160, 1919.

3. Ollier, Louis: Traité experimental et clinique de la régénération des os et de la production artificielle du tissue osseux, Paris, 1, 1867.

4. Truesdell, E. D.: Inequality of the Lower Extremities Following Fracture of the Shaft of the Femur in Children, Ann. Surg. **74:**498 (Oct.) 1921.

5. Speed, K.: Longitudinal Overgrowth of Long Bones, Surg. Gynec. Obst. **36:**787 (June) 1923.

6. David, V. C.: Overgrowth Following Fractures, Arch. Surg. **9:**438 (Sept.) 1924.

Harbin [7] have all reported instances of the overgrowth of bone following fracture or irritation of the shaft. Of all these cases overgrowth of the epiphyses could be excluded in only one, which was reported by Speed. The patient was 45 years old, so that compensatory epiphyseal growth was out of the question. Increase in length of bone in tertiary syphilis is not an uncommon observation, and it seems to be fairly characteristic of Paget's disease. While enormous increase has frequently been noted in acromegaly, one can fairly question how frequently actual lengthening occurs in the shaft of the long bones after the epiphyses have closed. There is no doubt, however, about the increase in the length of the ribs and the mandible. McEwen's [8] well known case of resection of the humerus for osteomyelitis, with subsequent transplantation of bone is an oft-cited example of growth of longitudinal bone. The patient, then 3 years old, was operated on in 1878. This is probably the earliest successful bone graft on record. At the time of operation, a graft 4½ inches (11.2 cm.) long, taken without periosteum, was inserted into a large defect of the humerus. Thirty years later, the graft measured 6 inches (15.2 cm.). The picture, included in his book, "The Growth of Bone," is difficult to interpret and to me not altogether convincing.

Haas, employing essentially the same technic which we shall describe, demonstrated almost incontrovertibly that no longitudinal growth occurs in the shaft of the tibia of the dog or in the metatarsals of the rabbit.

Still a bit skeptical, thinking that perhaps the cancellous bone near the epiphyses, being softer and more readily absorbed, might possess the property of increasing in length, we placed, not two markers near the center of the shaft as has usually been done, but several, 1 cm. apart along its entire length, so that any change occurring in the diaphysis might be observed.

EXPERIMENTAL WORK

Seventy-nine experiments in all were performed on young rabbits. Thirty-nine rabbits died of exposure or epidemic, leaving forty from which deductions might be drawn.

Method.—Under ether anesthesia, employing careful aseptic technic, the lateral surface of the femur was exposed throughout its entire length. This was done by making an incision parallel to the femur on the lateral surface of the thigh. After the skin and fascia lata were divided, the vastus lateralis and intermedius were retracted anteriorly and the biceps femoris posteriorly. Then, after the

7. Harbin, M.: Non-Suppurative Osteomyelitis, J. Bone & Joint Surg. **8**:401 (April) 1926.

8. MacEwen, W.: The Growth of Bone, Glasgow, Maclehose, Jackson & Co., 1912.

lower margin of the insertion of the gluteal muscles had been cut a metal rule was placed parallel to the femur, and marks 1 cm. apart were made on the bone with a knife. A small notch was made over each mark, and in these notches holes were drilled, care being taken not to go completely through the cortex. The drill used was slightly smaller than the shot inserted, so that it was possible to inlay the shot snugly into the holes. The shot rarely entered the canal of the marrow. The attachment of the gluteal muscles, the fascia lata and the skin were approximated with catgut, and a dressing of collodion was applied. There was only slight temporary impairment in the function of the leg after operation. Roentgenograms were usually made the following day. All films were made with the leg of the animal held firmly against the plate and with a uniform target distance of 24 inches (60.9 cm.). The superimposability of the films attests to the accuracy of the method.

Five series of experiments were performed as follows:

Series 1.—Only three or four shot were inserted. An attempt to close the lower epiphyses was made by removing from 1 to 2 cm. of the distal periosteum, leaving the articular cartilage adjacent to the outer margin of the epiphyseal plate intact.

Series 2.—From five to seven shot were inserted in the femur 1 cm. apart, care being taken not to disturb the periosteum or the blood supply to the epiphyseal plate. The end shot were placed close to the epiphyseal plates, and in rabbit 30 the lower shot was placed directly in the cartilage plate.

Series 3.—Shot were inserted in a manner similar to that used in the second series, except that an attempt was made to close the epiphyses by removing from 1 to 2 cm. of the distal periosteum, together with that part of the articular cartilage directly over the epiphyseal plate. Several parallel holes were drilled through the epiphyseal plate with a fine drill.

Series 4.—A procedure similar to that of series 3 was followed, with the exception that holes were not drilled in the epiphyseal cartilage plate.

Series 5.—Parallel holes were drilled in the epiphyseal plate, but no attempt was made to destroy its blood supply by removing the adjacent periosteum or articular cartilage.

It is evident from the following experiments that normally no growth of the diaphyses occurs in rabbits, except at the epiphyseal cartilage plate, and that if the shot is introduced carefully into the epiphyseal plate there is little or no retardation of growth (plate 30).

For the sake of brevity, only a few protocols are given and a few typical plates are shown.

Series 1, Rabbit 15.—The epiphyses were not completely closed. Three shot were inserted into the shaft of the left femur about 1 cm. apart. An attempt to close the epiphyses was made by destroying from 1 to 2 cm. of the distal periosteum, leaving the adjacent articular cartilage intact. Roentgenograms taken seven

and fifty-eight days after operation show no separation of the shot, but show an increase in the length of the diaphysis occurring proximal and distal to the shot.

Series 2, Rabbit 30.—The epiphyses were not closed. Seven shot were inserted in the shaft of the left femur about 1 cm. apart. The epiphysis with its blood supply was not disturbed. Roentgenograms taken three, fifty-two and eighty-four days after the operation show no separation of the shot, but show an increase in the length of the diaphysis occurring proximal and distal to the shot.

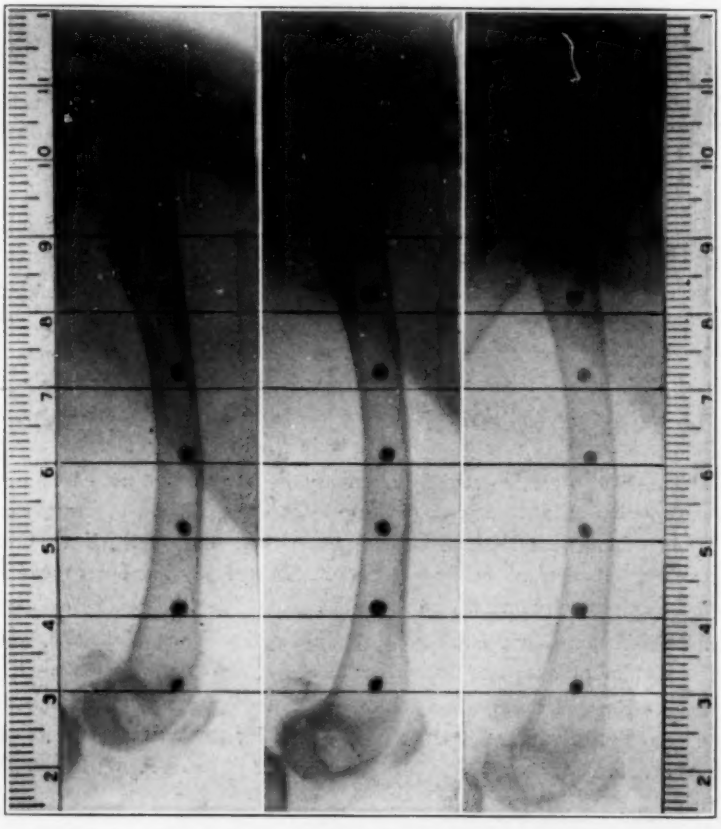

Fig. 1.—Roentgenograms of rabbit 30 taken three days, fifty-two days and eighty-four days after operation. The marker is in the epiphyseal cartilage plate. The epiphyses were open, and there was no separation of the shot.

It is also evident that when an increased functional demand is made by closure of the epiphyses no compensatory growth in the diaphysis occurs.

Series 3, Rabbit 39.—The epiphyses were closed. Six shot were inserted in the shaft of the left femur about 1 cm. apart. An attempt to close the epiphyses was made by destroying the periosteum with its blood supply and the articular

cartilage nearest the epiphyseal cartilage plate and by drilling holes parallel to and through the epiphyseal cartilage plate. Roentgenograms taken one, forty-seven and seventy-six days after operation show no separation of the shot, but a slight increase in the length of the shaft, occurring at both ends, but to a greater degree at the proximal end.

Closure of the epiphyses was not as readily accomplished in the rabbit as one might suppose. In some of his experimental work on dogs, Dr. Phemister had found that scraping the periosteum and peeling

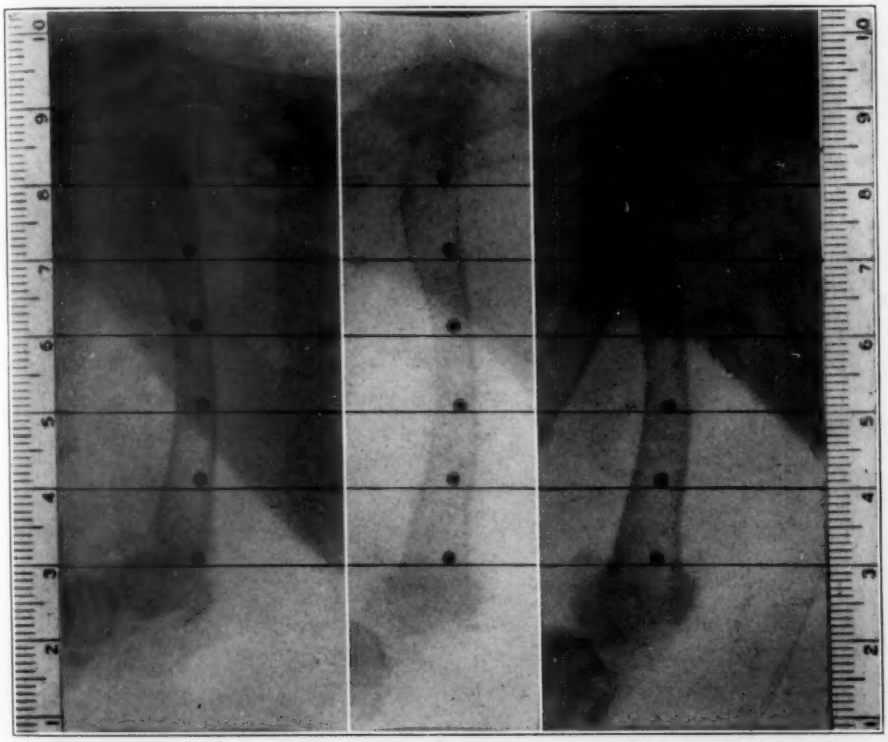

Fig. 2.—Roentgenograms of rabbit 39 taken one, fifty-two and eighty-four days after operation. The epiphyses were closed, and there was no separation of the shot.

the articular cartilage from the epiphyses was sufficient to cause cessation of growth. Haas,[9] however, working with rabbits, found that this procedure would hasten, but not immediately effect, closure of the epiphyses. In series 1, removal of the periosteum alone was performed with little success. In series 4, both the periosteum and the articular

9. Haas, S. L.: The Relation of the Blood Supply to the Longitudinal Growth of Bone, Am. J. Orth. Surg. **15**:15 and 305, 1917.

cartilage nearest the epiphyseal plate were removed, with evident retardation of growth, but in none was there immediate cessation of growth.

Series 4, Rabbit 60.—The epiphyses were closed. Six shot were inserted in the shaft of the left femur about 1 cm. apart. An attempt to close the epiphyses was made by destroying the periosteum with its blood supply and the articular cartilage nearest the epiphyseal plate. Roentgenograms taken three days and forty-four days after operation showed no separation of the shot, but a definite increase in the length of the diaphyses occurring both distal and proximal to the shot.

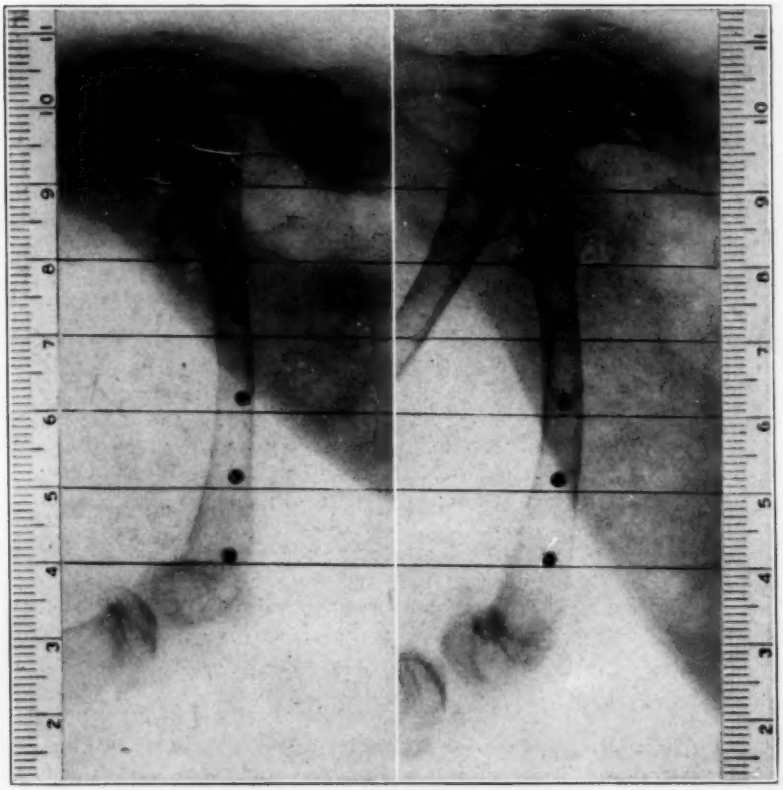

Fig. 3.—Roentgenograms of rabbit 60 taken three and forty-four days after operation. The epiphyses were open, and there was no separation of shot.

In series 5, an attempt was made to destroy the cartilage plate by drilling holes parallel to and through the plate. From five to seven holes drilled in this manner were sufficient in most cases to close the epiphyses, provided that the holes were drilled near the distal sides of the plate. This procedure seemed to be just as effective alone as when used together with scraping of the periosteum and articular cartilage as in series 3.

Series 5, Rabbit 70.—The epiphyses were closed. Six shot were inserted in the shaft of the left femur about 1 cm. apart. An attempt to close the epiphyses was made by drilling holes parallel to and through the epiphyseal plate. Roentgenograms taken three and thirty days after operation showed no separation of the shot, but a slight increase in the length of the diaphyses occurring both distal and proximal to the shot.

CONCLUSIONS

From these experiments it seems evident that in rabbits:

1. No longitudinal growth occurs in the diaphyses of long bones except at the epiphyseal cartilage plates, either under normal conditions,

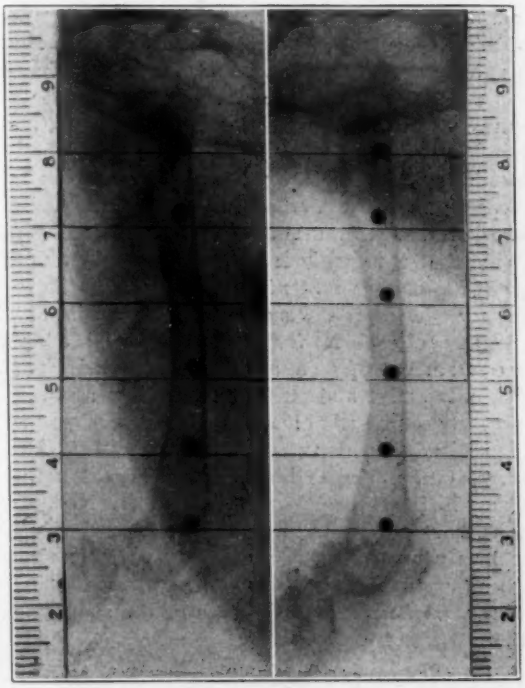

Fig. 4.—Roentgenograms of rabbit 70 taken three and thirty days after operation. The epiphyses were closed, and there was no separation of the shot.

or when an increased functional demand is made by closing or hastening the closure of the epiphyses.

2. Destruction of the extrinsic blood supply to the epiphyses by removing the adjacent periosteum and articular cartilage is not sufficient to close the epiphyses immediately, but will hasten the closure.

3. Immediate closure of the epiphyses can be accomplished by destroying the epiphyseal cartilage plate on the side nearest the joint.

PARAPLEGIA ASSOCIATED WITH CONGENITAL SCOLIOSIS *

REPORT OF A CASE

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WITH COMMENTS BY CLARENCE L. STARR
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History.—During December, 1925, a youth, aged 18, was admitted to the Toronto General Hospital. His legs were almost completely paralyzed, and there was a marked curvature of the spine. After a careful study and consideration of the history and roentgenograms it was believed that the spinal curvature was not of tuberculous origin. The case immediately attracted special interest, since no one on the surgical staffs of either the General or the Sick Children's Hospitals could recall having seen a case of paraplegia due to congenital scoliosis.

A case reported by Sachs encouraged us in our opinion that the paralysis was caused directly by the deformity of the spinal canal, and not by some separate pathologic process, such as a tumor.

The patient said that apparently his back was straight until he was 7 or 8 years of age, when his mother first noticed a slight curvature. At the age of 10 or 11, the deformity became obvious. At that time he developed the habit of leaning against any solid object for support, to ease his back, but at no time was there any great amount of pain, although in the mornings his back would ache slightly. He was active and was a good runner and bicycle rider. Two years prior to presentation he ran errands for a butcher, and after carrying the heavy baskets, he noticed that his right side was tired at night. About one year later, a gradual onset of weakness in the right leg was noticed, and he often tripped. A few months later, the other leg became involved, and he was unable to walk a block without getting tired, but he could still ride his bicycle. The weakness of the legs progressed gradually and was most marked in the right leg. Two weeks prior to presentation, it was practically impossible for him to walk; numbness was noticed over the base of the spine and the shin of the right leg, and there was some incontinence of urine.

Examination.—An examination of the patient on admission to the hospital showed that both legs were spastic, especially the right, and that they could be raised and lowered slowly. The reflexes at both knees and ankles were markedly exaggerated. There was a marked bilateral ankle clonus and positive Babinski sign. Areas in which sensation to pin-prick was dull were scattered over both legs, and at various points the patient showed inability to differentiate heat from cold.

The patient was put on a Bradford frame with efficient extension of the spine for two months, but despite this, the paraplegia and sensory loss slowly progressed.

Two months after admission an examination showed that there was no voluntary movement of the right thigh, leg, foot or toes. There was slight flexion of the left knee with some movement of the toes. The reflexes were markedly exaggerated at the knee and ankle, with bilateral ankle clonus and positive

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Babinski signs. The sensory changes which were present at the time are shown in figure 1. There was incontinence of urine. A lumbar puncture was performed with the Ayer manometer, and the spinal fluid showed an initial pressure of 25 cm. of fluid. There was no response on jugular compression. The fluid showed no tinge of yellow; the Pandy test was strongly positive with 1 drop; the Ross-Jones test was also strongly positive. The total protein was not estimated. It was obvious from the examination that the patient had a complete block of the spinal subarachnoid space, and that the paraplegia was rapidly progressing.

The motor disturbance was most noticeable in the right leg. Inability to differentiate heat from cold was most marked on the left. Sensation to light touch with wool and a pin scrape was dull on both sides, but was better appre-

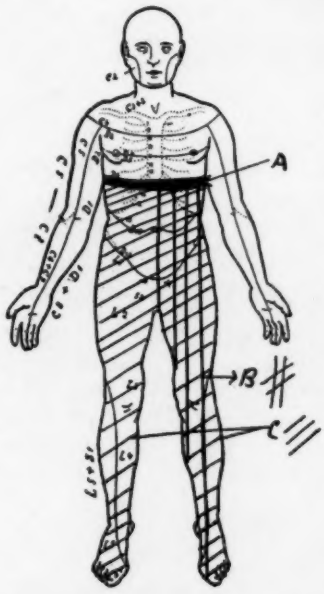

Fig. 1.—Sensory changes present two months after the patient was admitted to the hospital. *A* indicates the band of hyperalgesia. *B*, the area in which there was inability to differentiate heat from cold. *C*, the area in which sensation to pinscrape was dull; it was sharper on the left side. Sensation to light touch was dull over both legs, but more acute over the left.

ciated on the left. These observations suggested that the greatest interference with function was in the right half of the spinal cord.

Iodized oil was injected by the cisternal route, and a metal marker placed on the skin. A roentgenogram, taken after the patient had been sitting up for fifteen minutes, showed that the iodized oil was arrested at the point of maximum curvature, and definitely demonstrated the situation of the block in the subarachnoid space (fig. 2).

Operation and Course.—April 13: Operation was performed under ether anesthesia. The marker on the skin enabled one to plan the incision accurately. A moderate amount of difficulty was encountered in uncovering the laminae, retraction of the left scapula being necessary. When the canal was opened it was seen

to be filled with a vascular, fatty tissue. On clearing away this fat, it was apparent that the canal was large, with the comparatively small dural sac lying tightly against the bone on the inner side of the curve (fig. 3). Two roots were cut, which were tightly stretched in their dural sheaths. The dura was then opened, care being taken to keep the arachnoid intact. The cord immediately bulged out so that the edges of the dura were separated by at least 1 cm. (fig. 4). There was no apparent subarachnoid space over the cord except in the lower and upper parts of the field, where the cord was covered with a thin layer of fluid, that is, the point of maximum constriction of the cord by the dura was in the center of the field. At this point the vessels on the cord were barely

Fig. 2.—Exact situation of the block in the subarachnoid space, as shown by a cisternal injection of iodized oil, is indicated by the lower arrow. The skin marker is seen in the upper part of the figure.

apparent, but they were visible above and below. The dura was left open; in fact, there was no other choice. The subarachnoid space over the surface of the cord, at the point of maximum curvature, showed no signs of filling when closure was commenced. There was no evidence of the iodized oil in the subarachnoid space above, but, as the patient was on his face, this would be on the under surface of the cord, and not visible. There was no evidence of inflammatory reaction from the iodized oil, which had been injected two weeks previously. After all bleeding had been carefully controlled closure of the muscle was made with three layers of interrupted chromic catgut sutures.

It was satisfactory to have been able to preserve the arachnoid intact. Because of this, it was felt that a leak of cerebrospinal fluid would be unlikely,

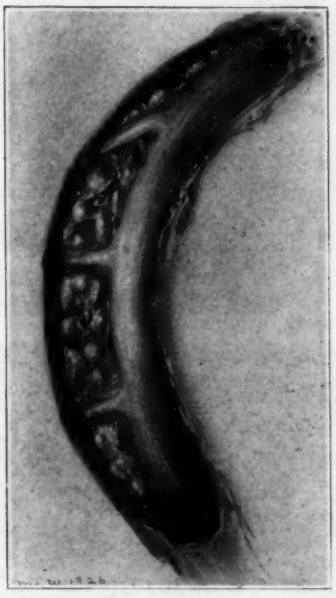

Fig. 3.—Marked enlargement of the bony canal on the outer side of the curve. On the inner side of the curve the dura is seen lying tightly against the bone. The dural sac is obviously much smaller at the point of maximum curvature.

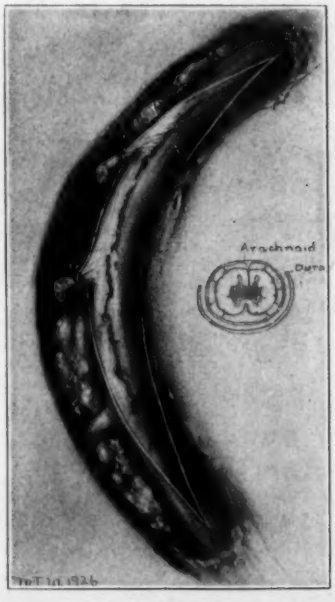

Fig. 4.—Bulging of the cord after the dura was opened. The cross-section gives a more definite impression of the extent to which the cord bulged through the opened dura; the arachnoid was kept intact.

and that adhesions would not form between the cord and the overlying muscle. The chief concern was lest the dura had not been opened over a sufficiently wide area to relieve pressure on the cord completely.

April 14: The patient was able to move his left ankle, whereas before operation he could barely move his big toe.

April 15: The patient moved both ankles rather freely and felt pinching over both thighs. There was control of the bladder.

Progress continued to be rapid. The patient was fitted with a spinal jacket, and was allowed to walk in six weeks. A roentgenogram taken at this time (fig. 5), showed the iodized oil at the bottom of the canal.

Fig. 5.—Six weeks after the operation. Most of the iodized oil is at the bottom of the spinal canal. The two silver clips in the upper part of the figure were left in to mark the point at which the cord appeared to be most constricted.

The patient was examined by the surgical staff of the Toronto General Hospital, Jan. 15, 1927, nine months after his operation. There was excellent power in both legs, so that he was able to stand with ease on his tiptoes, and readily climbed on to a table. There were no subjective or objective sensory changes. The function of the bladder apparently was normal. The reflexes were still exaggerated at the knees, but he did not have ankle clonus, and response to plantar stimulation was normal.

COMMENT

By Clarence L. Starr

The case presented by Dr. McKenzie has many points of interest, not least of which is the salvage of this patient from the derelict group. I believe that he would never have recovered by any other than operative means. His recovery is complete so far as his nervous system is concerned, and since the deformity appears fixed, it is unlikely that the paraplegia will recur.

Fig. 6.—A photograph taken on discharge from hospital, showing the deformity of the back.

The diagnosis is the important factor in all of these cases with paraplegia.

In Pott's disease one frequently sees paraplegia occurring in some stage of the disease. This is never due to bony narrowing but generally to thickening of the meninges, owing to the extension of the tuberculous disease from the bony focus. Occasionally it may be due to the presence of an abscess, or, more rarely, to a portion of bone sequestrated from the vertebral body or pedicle.

In all autopsy specimens ample space is shown in the neural canal, although the cord is shown displaced toward the front of the canal. This is found to be true also in cases in which operation is performed.

In childhood or adolescence, extradural fat is found diffusely scattered about the cord, apparently filling up the unoccupied space. Operative measures are for the most part unnecessary in these cases as nearly all patients recover with fixation or traction.

In structural or idiopathic scoliosis, even when the deformity has advanced to an extreme degree, paraplegia must be extremely rare as no cases have come under my observation.

In scoliosis following poliomyelitis, even when the deformity is great, paraplegia must be exceedingly rare as no cases have been seen in our clinic. Elmslie [1] reports one case with high grade deformity at the level of the fifth dorsal segment, due to poliomyelitis contracted at 7 years of age. The scoliosis developed when the patient was 8 years old; a spastic paraplegia commenced at 16 and progressed so that she was unable to walk at 17. She had anesthesia from the xiphoid down. Iodized oil injected into the cisterna magna showed a complete block at the sixth dorsal vertebra. Laminectomy was performed, and the dura was found stretched over the deformity, which was opened and left open. Muscle fascia was closed over the canal. The patient improved, but never to the extent shown in the case reported by Dr. McKenzie. At the end of one year, voluntary movements of the legs were possible, and she was beginning to make an effort to walk. Collier [2] reports a similar case which he believed was due to poliomyelitis. Scoliosis was noted at 4 years of age. At 14, the legs began to drag, and at 15, she had loss of sphincter control and spastic paraplegia. Sensation was diminished from the seventh dorsal vertebra down. An operation was not performed, but Mr. Collier suggested that decompression was indicated if no improvement was noted after rest and extension.

Five cases of congenital scoliosis with late symptoms of paralysis have been found in the literature, besides the case reported by Dr. McKenzie. Collier [2] reports one patient with a congenital defect in the upper dorsal vertebrae who first showed signs of weakness at the age of 10. There was progressive weakness with spasticity and exaggerated reflexes, including loss of bladder control. Iodized oil injected into the cisterna magna showed a block at the third dorsal vertebra. Treatment was not instituted.

Kleinberg [3] reports a case of congenital scoliosis with marked deformity in a girl, aged 7. At the age of 7 years and 6 months she

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1. Elmslie, R. C.: Two Cases of Scoliosis with Paraplegia, Proc. Roy. Soc. Med., Sec. Orth., no. 8, **18**:25 (June) 1925.

2. Collier, James: Case of Paraplegia in Scoliosis, Proc. Roy. Soc. Med., Sec. Neurol. no. 9, **18**:8 (Feb.) 1925.

3. Kleinberg, S.: Structural Scoliosis Complicated by Paralysis of the Lower Limbs. Report of a Case, J. Bone & Joint Surg. **5**:104, 1923.

rapidly developed a spastic paraplegia. The patient improved when placed in a recumbent position on a plaster bed and with extension applied. Ultimately she was able to walk freely.

Sachs [4] reports a case, apparently of congenital scoliosis with paraplegia, in which the patient recovered following operation.

Jaroschy [5] reports a case from the Biede Clinic of Prague. A patient, who had a severe deformity in the cervical spine in early childhood, rapidly developed paraplegia at the age of 18 and was finally completely bedridden. Roentgenograms showed a wedge-shaped half vertebra at the fourth dorsal and only eleven ribs were found on the right side. Neurologic examination revealed the usual spastic paraplegia with exaggerated reflexes and sensory disturbance. Laminectomy showed a good deal of epidural fat with apparent stretching of the dura over the prominence. The dura was not closed, and the muscles and fascia were closed over the cord. Ten weeks after operation the patient showed marked improvement. He could walk with two sticks, the spastic condition had disappeared, and the reflexes were normal. Sensation was almost normal.

A second case is reported in a girl, aged 14, who had deformity at the age of 2, but no signs of weakness developed until seven years later. At the time of examination, a roentgenogram showed a congenital defect similar to that in the first case and spastic paraplegia with exaggerated reflexes. There was no sensory or sphincter disturbance. Treatment by extension for three weeks did not cause any change in the condition, and a laminectomy was performed. The dura was not unduly tight, but it was opened and left open. Seven months after operation, the patient was able to get out of bed greatly improved. An examination one year after operation showed that she was able to walk freely. A slight increase in reflexes was the only pathologic symptom.

Ridlon [6] reports two cases, one apparently of congenital scoliosis with paraplegia in which the patient was placed in a recumbent position on a hyperextended frame. At the end of three years the reflexes were much improved but still overactive. Sensory reactions were normal. The second case was apparently one of spina bifida, and the paralytic symptoms were obviously due to the congenital defect of the cord.

The case presented by Dr. McKenzie is, I believe, one of congenital defect in the bony spine, and is the only one I have seen accompanied by

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4. Sachs, Ernest: An Unusual Case of Paraplegia Associated with Marked Gibbus and a Localized Collection of Fat at the Site of the Gibbus, J. Bone & Joint Surg. **7**:709, 1925.

5. Jaroschy, W.: Delayed Lesion of Spinal Cord from Congenital Scoliosis, Beitr. z. klin. Chir. **129**:348, 1923; abstr. J. A. M. A. **81**:615 (Aug. 8) 1923.

6. Ridlon, John: Report of Two Cases of Scioliosis, J. A. M. A. **67**:803 (Sept. 9) 1916.

complete paraplegia. The extreme deformity without paralytic onset, the early appearance of deformity and the obvious segmentation of several of the upper dorsal vertebrae point to the congenital origin. Clinical or roentgen-ray signs of abscess were not noted, and evidence of tuberculous disease was not present in the neural canal at operation.

In my opinion, the extradural fat found at operation, as in the case reported by Sachs, was not a factor in the causation of the paraplegia.

The rapid restoration of the form of the cord from the flattened shape to the normal rounded appearance and the commencement of pulsation after the opening of the dura seem to prove conclusively that the cause of the paraplegia was the pressure of the cord against the acute bony angle, by the taut dura.

FATAL PULMONARY EMBOLISM

A STATISTICAL REVIEW

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Records of postmortem examinations at the Mayo Clinic for the last ten years show that pulmonary embolism occurred in 313 cases; in 267 of these, the complication followed operation and in 46, it occurred in nonsurgical cases. In 223 (83.5 per cent) of the 267 cases, emboli were the primary cause of death, and in 44 (16.5 per cent), they were either contributory factors or were so small that they had little or no connection with the patient's death.

One hundred and four of the patients who died from embolism were men and 119, women; their average age was 53.2 years. The average age of 1,000 adult patients operated on consecutively was found to be 42.8 years. Therefore, the average age of the patients dying from embolism was 10.4 years greater than that of the average patient coming to operation. The average height for these patients was 5 feet, 6.5 inches (168.9 cm.), and the average weight, 168 pounds (76.2 Kg.). The normal weight for persons of this age and height has been estimated at 155 pounds (70.3 Kg.), so these patients averaged 13 pounds (5.9 Kg.) overweight. The fact that most of them were ill when they came to the clinic and definitely below their usual weight must also be taken into consideration. The average systolic blood pressure was 138 and the average diastolic blood pressure, 82, which is a normal or somewhat subnormal blood pressure for patients of this age. That massive pulmonary embolism may occur in hypertension is shown by the fact that the systolic blood pressure was more than 200 in five cases in the series, the highest pressure encountered being 260.

The operation leading to fatal embolism in this series was extra-abdominal in 43 cases (19.3 per cent) and intra-abdominal in 180 (80.7 per cent). Forty-four per cent of the operations within the abdomen were performed in the upper portion and 51.6 per cent in the lower portion; 3.7 per cent were explorations and 0.5 per cent, paracenteses. In this series, therefore, there was no great difference in the number of deaths from pulmonary embolism following operations in the upper and in the lower portion of the abdomen.

A more detailed review of the cases of embolism occurring after intra-abdominal operations (table 1) shows that the incidence of fatal cases following operations on the gallbladder and ducts was 0.3 per cent; on the stomach, 0.25 per cent; on the uterus and its appendages, 0.38 per

cent; on the prostate, 0.46 per cent and on the bladder (cystostomy preliminary to prostatectomy), 1.5 per cent. That the incidence of embolism is higher after cystostomy alone than after prostatectomy is probably accounted for by the fact that the patients undergoing cystostomy were such poor risks that they were not subjected to the one-stage operation.

TABLE 1.—*Pulmonary Embolism Following Intra-Abdominal Operations*

Operations on	Total Number of Operations, 1917-1926	Cases of Embolism		Incidence of Embolism	
		Number	Percentage	All Cases, Percentage	Fatal Cases, Percentage
Gallbladder and ducts	11,689	37	17.2	0.31	0.30
Stomach	12,453	38	17.6	0.30	0.25
Uterus and appendages	11,961	51	23.7	0.42	0.38
Bladder:					
Cystostomy for hypertrophy of prostate	1,104	19	....	1.72	1.50
Prostatectomy	1,959	11	13.5	0.56	0.46
Operations other than simple cystostomy	922	5	2.3	0.54	0.54
Colon	2,389	20	9.0	0.83	0.48
Abdomen:					
Exploration	.....	8	3.7	....	....
Paracentesis	321	1	0.5	0.30	0.30
Appendix	12,356	5	2.3	0.04	0.02
Kidneys	3,635	5	2.3	0.10	0.10
Rectum	1,982	5	2.3	0.25	0.25
Spleen	317	4	1.9	1.26	0.90
Subdiaphragmatic abscess	29	1	0.5	3.40	3.40
Omentum (tumor)	2	1	0.5	50.00	50.00
Small bowel	991	3	1.4	0.30	0.30
Ventral hernia	1,235	4	1.8	0.30	0.30

TABLE 2.—*Cases of Fatal Embolism Resulting from Extra-Abdominal Operations*

Region of Operation	Cases
Hernia	14
Breast	8
Spinal cord	3
Long bones (reduction of fracture)	3
Long bones (open operation)	2
Chest (aspiration)	2
Minor gynecology	2
Trifacial nerve (avulsion for neuralgia)	1
Brain	1
Spine	1
Thyroid	1
Axillary glands	1
Face (plastic operation)	1
Rib (resection)	1
Arm (amputation)	1
Left hip (operation for tumor)	1

In the forty-three cases in which fatal embolism occurred following extra-abdominal operations, herniotomy was performed in fourteen, operation on the breast in eight, on the spinal cord in three, and on the long bones in two, aspiration of the chest in two and the reduction of fracture of long bones in three (table 2).

The deaths in the series of fatal cases of embolism represent 6 per cent of all deaths following operations during the last ten years. On the other hand, in a complete analysis of deaths resulting from opera-

tions during 1925, one period was found in which 2,606 consecutive operations were performed without a single proved case of pulmonary embolism. These operations did not include operations on the nose and throat, hemorrhoidectomy, aspiration, transfusion or similar surgical procedures.

Definite postoperative infections, such as infected wounds, pyelonephritis, miscellaneous abscesses, acute pericarditis or peritonitis, had occurred in ninety-nine cases (44 per cent of the series). It should be remembered that these infectious conditions brought about the added complication of a more prolonged convalescence in bed for the patients. Twenty-nine of them had postoperative bronchitis and twenty-eight had postoperative phlebitis or symptoms suggestive of this condition. The average interval between the date of operation and the date of death was fourteen days. In sixty-nine cases, death was described as occurring suddenly; in thirty-four others it occurred within ten minutes of the first seizure; in twenty-two, within thirty minutes; in ten, within one hour; in fifteen, within twelve hours; in twenty-five, within twenty-four hours; in twenty-three, within seven days, and in six, within fourteen days; in nineteen, the time of seizure could not be established.

Obstruction of one of the large branches of the pulmonary artery is, of course, a swift and hopeless tragedy for those patients in whom it is followed by death. Patients who are able to recover partially, however, from the shock caused by a large embolus, only to die from failing circulation if left unaided, have a chance to be treated by supportive and sometimes effective therapeutic measures. The possibility of saving the patient's life by an emergency operation, that is, removal of the clot, must be considered. This has been carried out successfully by Kirschner.[1] Two patients with unilateral pulmonary embolism have recently been treated successfully in the clinic by prompt administration of oxygen with the Barach-Roth tent. The subjective relief was almost immediate in both cases. Unfortunately, many patients who successfully withstand the first attack of embolism succumb to the second attack or to secondary pulmonary suppuration or pneumonia.

In fifty-six cases, forty-four surgical and twelve medical, although embolism was found at necropsy, it was not regarded as the principal cause of death. In most of these, none of the usual clinical signs of embolism had manifested themselves, and the condition was not suspected. In most instances death was due to gradual cardiac failure from myocardial degeneration, peritonitis, pneumonia or hemorrhage. The emboli that were found were mostly small and apparently came into the picture only as a terminal event.

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1. Kirschner, M.: Ein durch die Trendelenburgsche Operation geheilter Fall von Emboli der Arteria pulmonalis, Arch. f. klin. Chir. **133**:312, 1924.

In the nonsurgical group of forty-six cases (table 3), three patients with adenomatous goiter with hyperthyroidism and one with exophthalmic goiter, all of whom had myocardial degeneration, died from cardiac failure with decompensation. Nineteen other cases in this group were primarily cardiovascular, sixteen of them with marked decompensation. In half of the group of nonsurgical cases, therefore, death was accompanied by gradual cardiac failure. Although the emboli in many of these cases were small and relatively insignificant in their effects, their occurrence again emphasizes the importance of the condition of the circulatory system in bringing about thrombosis and embolism. It should be remembered that some of these apparent emboli may have been formed in situ.

TABLE 3.—*Deaths from Pulmonary Embolism in Nonsurgical Cases*

Cause	Deaths
Hyperthyroidism with adenomatous goiter	3
Exophthalmic goiter	1
Cardiovascular disease	19
Carcinoma of stomach	3
Carcinoma of ovary	3
Carcinoma of prostate	1
Carcinoma of uterus	1
Carcinoma of rectum	1
Cyst adenoma of ovary	1
Fibromyoma of uterus	1
Retroperitoneal lymphosarcoma	1
Retroperitoneal tumor	1
Pyelonephrosis	2
Hypertrophy of prostate	1
Fractures, multiple	1
Gangrene of leg	1
Cellulitis of face	1
Peritonitis	1
Pemphigus	1
Acute yellow atrophy of liver	1
Sciatica	1

The factors, other than slowing of the circulation, that are commonly regarded as concerned in the production of thrombosis are changes in the composition of the blood and injury to the vessel wall. The former has been considered by Allen [2] and Snell.[3] Thrombosis within veins that have been cut is a normal occurrence and necessary to the completion of any surgical procedure. This should extend within the vessel to the point of entrance of the next tributary vein; when it extends beyond this point into the larger vessels, a pathologic condition of grave peril is created, as portions of the clot may become detached in the blood stream. It is interesting to note that the source of emboli is often some distance from the operative site.

2. Allen, E. V.: Changes in the Blood Following Operation, Arch. Surg., published in this issue.

3. Snell, A. M.: The Relation of Obesity to Postoperative Pulmonary Embolism, Arch. Surg., published in this issue.

Thrombosis, which may have served as the possible source of emboli, was found in 189 of the total series of 313 cases. In some instances, two or even three of these sites of thrombosis were found in the same case. The four most common sources (table 4) were: the iliac vein in sixty-four cases, the femoral vein in fifty-five, the pelvic veins in forty-three and the prostatic plexus in eighteen. Seventy-nine per cent of the sites of thrombosis were in these situations. The groups in which the probable source of emboli was in the iliac or femoral veins were studied further. In 14 per cent of these cases, operation had not been performed; in 15 per cent, the operation had been extra-abdominal; in 25 per cent it had been performed in the upper part of the abdomen; in 46 per cent, in the lower part of the abdomen. In only one case had operation been performed on the lower extremity—open operation for

TABLE 4.—*Source of Emboli*

Source	Number
Iliac vein	64
Femoral vein	55
Pelvic veins	43
Prostatic plexus	18
Vena cava	13
Right auricle	10
Renal vein	7
Axillary vein	3
Right ventricle	3
Ovarian veins	3
Hemorrhoidal veins	1
Deep epigastric vein	1
Jugular vein	1
Vaginal plexus	1
Cervical plexus	1
Subclavian vein	1
Innominate vein	1
Azygos vein	1

fracture of the neck of the femur. Only two of the eighteen patients with thrombosis of the prostatic plexus had undergone prostatectomy. These statistics would indicate that the site of operation is not of paramount importance in determining the site of thrombosis and the source of emboli.

In only one case in the series was the probable source determined to be in the subclavian vein, and in three cases, in the axillary veins. Such factors as posture, stagnation of blood in dependent parts and decreased movement probably account for the more frequent occurrence of thrombosis in the veins of the pelvis and of the lower extremities than in the veins of the upper extremities. In order to obviate such factors, patients are encouraged to change their position often, and early passive and active movement of the extremities are instituted. Patients are encouraged to breathe deeply since the principal force causing blood to flow through the large abdominal veins is the negative pressure created in the chest during inspiration. In order to splint the abdomen

and facilitate deep breathing, patients who on account of age or obesity are regarded as being more susceptible to embolism are supplied with elastic abdominal belts. The support supplied by these belts when the patient has to cough is also gratifying.

Some previous impressions regarding pulmonary embolism have been shown by this review to be without foundation. One such impression was that there was probably a seasonal variation in the incidence, in other words, that there were more cases in the spring and autumn months. In this review, however, it was found that although there was some monthly variation during a single year, there was but little change from month to month when the period of ten years was considered as a whole.

SUMMARY

The incidence of fatal pulmonary embolism among the surgical cases that came to necropsy at the clinic during the last ten years is 6 per cent. Patients at the clinic who die from pulmonary embolism are older than the average surgical patient; they are somewhat overweight, and, as a group, have a normal or somewhat subnormal blood pressure; a high percentage have postoperative infections.

While the importance of the operative procedure in determining the site of thrombus formation and the occurrence of pulmonary embolism cannot be overlooked, other factors, such as age, weight, general condition of the patient, efficiency of the circulation, bodily inactivity incident to almost any operative procedure and infection, should also be emphasized.

THE RELATION OF OBESITY TO FATAL POST-OPERATIVE PULMONARY EMBOLISM *

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The present study of the causes of postoperative death of obese patients was undertaken to determine, if possible, the cause of the increase in mortality in such patients and what measures might be taken to reduce it. This involved a comparison of the causes of death of obese patients with those of a control group. The latter was made up of all patients who died following operation during the same period, without regard to weight.

BASIS OF STUDY

The history of the patient in all cases of death after operation during a period of six years (from 1920 to 1925, inclusive) was studied. A total of 156 of these patients were definitely obese. In 145 cases of this group, necropsy was performed; in the remaining eleven, the clinical cause of death seemed sufficiently clear to permit their inclusion in the group.

The obesity in these 156 cases was graded from 1 to 4: 1 indicated slight obesity (from 20 to 35 pounds [9.0 to 15.9 Kg.] overweight); 2, moderate (from 35 to 60 pounds [15.9 to 27.2 Kg.] overweight); 3, marked (from 60 to 100 pounds [27.2 to 45.4 Kg.] overweight), and 4, extreme (more than 100 pounds [45.4 Kg.] overweight). The cases were divided into four groups according to this grading. Standard actuarial tables of weight were used as a basis for the classification. The percentage of incidence of peritonitis, embolism, pneumonia and other diseases in each group was compared with the percentages of each of these complications in the whole series of 1,942 cases in which necropsy was performed.

As several possible causes may co-exist in the same person, the "principal cause of death" given on the death certificate was used as a basis for classification; consequently, the figures do not show the total incidence of peritonitis, pneumonia, and other diseases. The headings in table 1, which gives the causes of postoperative death of obese patients, require an explanation. All cases of fatal thrombosis of the abdominal, cerebral, and peripheral vessels are listed under "thrombosis." Cases of death from uremia, hemorrhage, operative shock and other causes are included under "miscellaneous." The "unexplained" deaths form a distinct clinical group and will not be discussed in detail.

* From the Division of Medicine, Mayo Clinic, and The Mayo Foundation.

Peritonitis, pneumonia and pulmonary embolism are the three major causes of postoperative death in the control group and in the group of obese patients. The experience of the Mayo Clinic, from 1920 to 1925, inclusive, was that these three causes accounted for 40 per cent of all postoperative deaths; pneumonia was the principal cause in 13.8 per cent; peritonitis, in 18.3 per cent and pulmonary embolism, in 7.9 per cent of all cases which came to necropsy. The figures for pneumonia and peritonitis are substantially the same in the group of obese patients as in the control group; pulmonary embolism, however, was about three times as frequent a cause of death in the former as in the latter. Embolism accounted for death in 20 per cent of the fatal cases in groups 1 and 2 (patients from 20 to 60 pounds overweight) and in

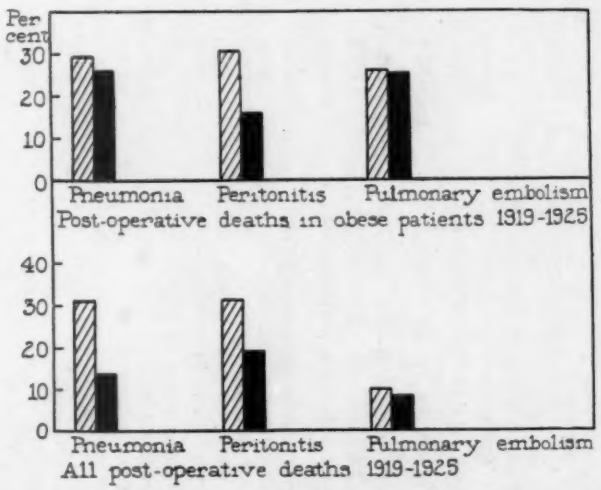
TABLE 1.—*Causes of Postoperative Death in Obese Persons*

		Peritonitis		Pneumonia		Embolism		Thrombosis		Sepsis		Jaundice		Unexplained		Miscellaneous	
Group	Cases	Cases	Percentage	Cases	Percentage	Cases	Percentage	Cases	Percentage	Cases	Percentage	Cases	Percentage	Cases	Percentage	Cases	Percentage
Group 1: slight obesity (from 20 to 35 pounds overweight)	40	7	17.5	4	10.0	10	25.0	1	2.5	4	10.0	3	7.5	..	....	11	27.5
Group 2: moderate obesity (from 35 to 60 pounds overweight)	62	13	21.0	10	16.2	12	19.3	5	8.1	7	11.3	..	....	1	1.6	14	22.6
Group 3: marked obesity (from 60 to 100 pounds overweight)	47	9	19.2	2	4.2	16	34.0	2	4.2	4	8.5	1	2.1	7	14.9	6	12.8
Group 4: extreme obesity (100 pounds or more overweight)	7	4	57.0	..	....	2	28.6	..	....	..	....	..	....	..	....	1	14.3
Total	156	33	21.2	16	10.3	40	25.6	8	5.1	15	9.6	4	2.6	8	5.1	32	20.6

about 30 per cent in groups 3 and 4 (patients more than 60 pounds overweight). Thrombosis of cerebral and abdominal vessels (a relatively infrequent cause of postoperative death) was found in 5 per cent of the obese patients. The percentage of incidence of the major causes of death in the group of obese patients is contrasted graphically in chart 1 with that in the control group.

The cases were analyzed in detail in order to determine any possible causes for this remarkably high incidence of fatal thrombosis and embolism in patients who were overweight. The significant observations on the forty obese patients who died from pulmonary embolism are summarized in table 2. The patients were divided into four groups similar to those in table 1. Twenty-two of the patients were women and eighteen were men, the average age of the former being 53 and of the latter, 56. In spite of the rather advanced age of the patients, clinical evidence of degenerative cardiac, vascular or renal changes was rather

infrequent. The blood pressure was usually within normal limits: in four cases the diastolic pressure was more than 100 mm.; in seven, the systolic pressure was more than 160 mm. In one case, cardiac enlargement was graded 2, in another there were significant electrocardiographic changes (T wave negativity in derivations I, II, and III); in both instances the blood pressure was normal. Definite evidence of renal insufficiency was not presented in any case. Phlebitis was noted as a postoperative complication in five cases. The weight had been reduced prior to operation in only two cases and in one of these 37 pounds (16.8 Kg.) had been lost by active reduction of weight in the hospital; in the other, 30 pounds (13.6 Kg.) had been lost in two years at home. About half of the cases of fatal pulmonary embolism in this



Percentage of incidence of the major cause of death in obese patients and in the control group. The black columns represent cases in which the designated condition was the major cause of death; the shaded column, cases in which the condition was reported present.

group followed operations on the pelvic organs and lower urinary tract. This does not necessarily imply that these particular types of operation increase the liability to embolism, since the factor of age cannot be eliminated; prostatectomy, hysterectomy and related surgical procedures are usually performed on older patients. Ether was the anesthetic used in nineteen cases, procaine hydrochloride with or without a general anesthetic was used in eleven, and either nitrous oxide or ethylene, in seven. This is in accord with the average frequency of employment of these anesthetics at the clinic; consequently, anesthesia could hardly be considered a factor. Johnson,[1] who recently reviewed the experience

1. Johnson, A. C.: Unpublished data.

TABLE 2.—*Analysis of Cases of Fatal Pulmonary Embolism*

Case	Sex	Age, Years	Weight, Pounds	Blood Pressure		Operation	Anesthetic	Phlebitis	Days After Operation	Manner of Death	Reduction of Weight before Operation
				Systolic	Diastolic						
Group 1—Patients from 20 to 35 Pounds Overweight											
1	♀	51	160	158	76	Removal of carcinoma of right breast	No record	None	22	Gradual, from repeated embolism	None
2	♀	60	167.5	140	80	Exploratory (inoperable ovarian tumor)	Procaine (local) nitrogen monoxid	None	3	Sudden	None
3	♀	33	153	108	74	Left nephrectomy	Ether	None	8	Sudden	None
4	♂	60	185	190	80	Suprapubic cystostomy	Ether	None	22	Gradual, from multiple septic infarcts	None
5	♀	58	172	144	98	Suprapubic cystostomy for carcinoma of bladder	Ether	Present	28	Sudden	None
6	♂	53	187	128	80	Entero-enterostomy for carcinoma of bowel	Ether	None	9	Sudden	None
7	♂	50	198	105	66	Suprapubic cystostomy	Ether	None	5	Sudden	None
8	♀	40	158	170	110	Cholecystectomy and appendectomy	Ether	None	11	Sudden	None
9	♀	49	171	140	96	Laminectomy for tumor of the cord	Ether	None	3	Sudden	None
10	♂	71	198	136	89	Colostomy for carcinoma of rectum	Ether	None	21	Sudden	None
Group 2—Patients from 35 to 60 Pounds Overweight											
11	♀	67	170	146	74	Vaginal hysterectomy	No record	None	13	Gradual	None
12	♂	63	202	160	90	Suprapubic prostatectomy	Ether	None	18	Sudden	None
13	♂	55	220	140	90	Suprapubic prostatectomy	Procaine (spinal)	None	15	Sudden	None
14	♀	53	190	160	90	Repair of large umbilical hernia	Procaine	None	6	Sudden	None
15	♂	64	210	148	98	Suprapubic prostatectomy	Procaine	None	7	Gradual	None
16	♂	70	194	182	110	Suprapubic cystostomy	Procaine	None	11	Gradual, from repeated embolism	None

17	♀	56	210	152	80	Total hysterectomy	Ether	None	15	Sudden	None
18	♂	46	208	116	70	Cholecystostomy	Ether	None	3	Sudden	None
19	♀	56	180	140	88	Cholecystectomy and appendectomy	Ether	None	14	Sudden	None
20	♂	65	190	124	78	Repair of ventral hernia	No record	None	8	Sudden	None
21	♂	64	223	170	80	Gastro-enterostomy	Ether	None	9	Sudden	None
22	♀	57	200	150	70	Vaginal myomectomy	Ethylene and ether	Present	14	Sudden	None
						Group 3—Patients from 60 to 100 Pounds Overweight					
23	♀	60	226	150	90	Right inguinal herniotomy	Procaine	None	10	Gradual, from repeated embolism	10 pounds
24	♀	43	230	148	90	Cesarean section	Procaine, nitrogen monoxide	None	25	Sudden	None
25	♀	58	222	170	90	Hysterectomy	Ether	None	23	Sudden	Recent gain in weight
26	♂	44	245	120	80	Thyroidectomy	Procaine, ether	None	3	Sudden	None
27	♂	45	207	140	88	Cholecystectomy	Ether	None	10	Sudden	None
28	♀	44	209	125	85	Salpingectomy and oöphorectomy	Nitrogen monoxide and ethylene	None	5	Sudden	None
29	♀	74	198	104	78	Exploration of carcinoma of uterus	No record	None	9	Sudden	None
30	♂	78	225	180	70	Suprapubic cystostomy	Procaine	None	44	Gradual, from repeated embolism	None
31	♀	57	222	210	124	Laminectomy for tumor of cord	Procaine	None	3	Sudden	None
32	♀	50	185	180	110	Cholecystectomy	Ether	None	7	Sudden	None
33	♀	56	252	155	85	Subtotal hysterectomy	Ether	Present	30	Sudden	None
34	♂	43	254	138	95	Repair of inguinal hernia	Nitrogen monoxide and ether	None	4	Gradual	37 pounds (active reduction)
35	♂	46	230	124	102	Puncture of degenerating cerebral glioma	Procaine	None	6	.....	None
36	♂	49	232	140	82	Kraske operation for carcinoma of rectum	Procaine	Present	50	Sudden	None
37	♀	49	190	138	90	Total hysterectomy	Ether	None	5	Gradual	None
38	♀	60	205	150	88	Carcinoma of cervix uteri, total hysterectomy	Ether	None	3	Gradual	None
						Group 4—Patients More Than 100 Pounds Overweight					
39	♂	39	288	120	88	Cholecystectomy	Ether	None	10	Sudden	None
40	♀	32	267	138	100	Thyroidectomy	Nitrogen monoxide	Present	8	Sudden	None

* In this and the following tables, ♂ indicates male, and ♀, female.

at the Mayo Clinic in this matter, failed to find any relation between the various types of anesthetic and the incidence of postoperative embolism. The present series is too small to permit of any conclusions in regard to seasonal incidence.

The duration of life after operation varied from three to fifty days, the average being about thirteen days. As might be expected, death was sudden in most cases (thirty); in five cases, gradual failure was noted, and in five, repeated pulmonary infarctions occurred. In the latter group the patients survived the operation from ten to twenty-two days, and died with clinical symptoms suggesting bronchopneumonia.

TABLE 3.—*Analysis of Cases of Fatal Postoperative Thrombosis*

Case	Sex	Age, Years	Weight, Pounds	Obesity, Degree	Anesthetic	Operation	Site of Thrombosis	Days after Operation	Manner of Death
1	♂	33	175	1	Ether	Choledochostomy	Portal vein	12	Sudden collapse after stormy postoperative course
2	♀	42	180	2	Ether	Excision of cancer of right breast	Cerebral artery	8	Gradual
3	♂	76	200	2	Procaine, ether	Gastro-enterostomy for carcinoma of stomach	Coronary artery, pulmonary artery	9	Sudden
4	♀	40	205	2	Procaine, ethylene	Amputation of right arm for gangrene	Pulmonary, carotid and subclavian arteries	2	Gradual
5	♂	49	251	2	Ether	Right herniotomy	Mesenteric artery	3	Sudden
6	♀	42	218	3	Ether	Right temporal decompression for brain tumor	Superior longitudinal sinus	7	Sudden collapse after stormy course
7	♂	56	217	3	Ether	Splenectomy	Mesenteric vein	14	Gradual
8	♂	61	226	2	No record	Suprapubic prostatectomy	Inferior vena cava	120	Gradual

The relatively high frequency of fatal thrombosis is interesting although the factors that favor pulmonary embolism may not be operative in this group. Eight patients, approximately 5 per cent of the obese patients, died from this cause. The details of these cases are given in table 3. In one case, mesenteric thrombosis followed splenectomy; in another, thrombosis of the superior longitudinal sinus followed cranial decompression; in a third case, the presence of thrombosis of the subclavian artery had been determined before the arm was amputed. A fourth patient, aged 76, died from coronary thrombosis following operation. It is difficult to see how obesity could have had anything to do with the unfavorable outcome in these cases. The fact that in eight cases in the obesity group death was traced to thrombosis does not necessarily imply a relation between thrombosis and obesity.

CONCLUSIONS

The high incidence of pulmonary embolism as a cause of postoperative death in obese patients is of much interest. It suggests, but does not prove, that obesity, per se, increases the liability to this much feared complication. The average age in the group of 156 patients was 55; this emphasizes the relation between age and pulmonary embolism, previously noted by Lindsay [2] and Lister.[3] Difficulty of operation with unusual trauma may be a factor; mild circulatory failure with resultant venous stasis might conceivably be more common in obese persons. After operation on obese patients, there may be an increased liberation of thromboplastic lipoid substances such as kephalin, due to the extensive areas of fat invaded. True fat embolism, however, occurred in only two of the cases.

In interpretating the foregoing data one must bear in mind that there are no statistics available with regard to mortality and causes of death for particular operations according to age groups. These would serve as much more accurate controls than the whole groups of patients of all ages considered without respect to the type of operation. An analysis of the outcome of serious and extensive surgical procedures in older patients might well show a high incidence of fatal postoperative pulmonary embolism.

Observations on arterial and venous pressure and rate of circulation and studies of peripheral blood flow would probably shed much light on the subject of circulatory stasis, which is generally recognized as an important factor in the development of embolism. At the present time a definite regimen, designed to improve the general circulation and to combat venous stasis, is being tried at the Mayo Clinic under controlled conditions. Walters [4] has been interested in the use of thyroid extract in this connection, and has recently reported favorably on its use.

SUMMARY

Pulmonary embolism following operation seems to be a more common cause of death of the obese patient than of the average patient. Whether this is due to the age of the patient, to the type of operation performed, to unknown factors related to the flow and coagulation of blood or to the obesity, per se, cannot be definitely stated. From a

2. Lindsay, E. C.: The Prevention and Treatment of Postoperative Pulmonary Embolism, Lancet **1**:327, 1925.

3. Lister, W. A.: Causation of Pulmonary Embolism Following Operation, Lancet **1**:111, 1927.

4. Walters, Waltman: The Suggested Use of Thyroid Extract to Reduce the Incidence of Postoperative Embolism, Minnesota Med. **10**:25, 1927.

consideration of my statistics and those presented by Henderson,[5] it seems probable that there is a group of patients over 50 years of age, obese and with normal or subnormal blood pressure, who are particularly susceptible to pulmonary embolism as a postoperative complication.

5. Henderson, E. F.: Fatal Pulmonary Embolism: A Statistical Review, Arch. Surg., published in this issue.

POSTOPERATIVE PHLEBITIS

A CLINICAL STUDY *

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This study is based on eighty-seven cases of postoperative phlebitis that I have observed. Information and exact data were sought to determine the following points: (1) the minimal signs and symptoms on which to make a diagnosis of phlebitis; (2) the degree of disability consequent to this complication; (3) the etiology, predisposing factors, such as intercurrent infection, and seasonal incidence, and (4) the relationship, if such exists, of phlebitis to fatal postoperative embolism.

For a period of two years, I examined, as a routine, patients with pain, soreness or swelling in the legs following operation. The criteria for establishing a diagnosis of postoperative phlebitis were ill defined, and in many cases phlebitis was erroneously diagnosed, with an unmerited attendant increase in the postoperative convalescent period. Many patients complained of sensations in the legs and hips after operation, varying from fatigue and soreness to sharp twinges of pain. One group of patients complained of the symptoms that occurred on getting up from bed, usually from the seventh to the tenth day after operation. Other patients experienced soreness in the gluteal, calf or lumbar groups of muscles. Several complained of soreness in the feet from standing. In most instances, phlebitis was considered the trouble, and the patients returned to bed for an additional period of rest, with the leg elevated and heat applied. As some of these patients did not have phlebitis, it was obvious that minimal standards for the diagnosis were indicated. Two cases of septic thrombophlebitis were observed, but they are not included in this study.

MINIMAL DATA FOR THE DIAGNOSIS OF PHLEBITIS

The diagnosis of phlebitis in the legs was based on: (1) the situation of pain or soreness, (2) the presence or absence of edema and (3) the constitutional reactions. Tenderness along the course of the veins is the most important single sign of phlebitis, and the diagnosis was not made unless this symptom was present. A diagnosis was frequently made when edema and fever were absent; when present they are important corroborative signs. The presence of pulmonary infarction is significant, and occasionally directs attention to the possibility of phlebitis in the veins of the leg.

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* From the Division of Medicine, Mayo Clinic, and The Mayo Foundation.

The situation of the soreness in phlebitis is necessarily along the course of the vein affected (fig. 1). Three points or areas in the leg were examined for tenderness. One or more of these areas were tender on palpation in the definite cases of phlebitis. The middle of the calf, or the lower popliteal area, was tender on palpation in 64 per cent of the eighty-seven cases; in 9 per cent, the calf and lower and upper femoral areas were tender, and in 27 per cent, the upper and lower femoral areas. The right leg was affected in 16 per cent; the left, in 64 per cent and both legs, in 20 per cent of the cases. Tenderness or subjective soreness was confined closely to the vein in a longitudinal direction. Frequently the vein could be felt as a tender, hard cord. Often deep pressure

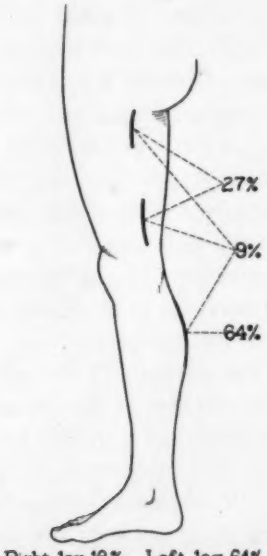

Fig. 1.—Points of tenderness in cases of postoperative phlebitis.

was necessary to elicit pain, and often the patient could accurately trace the course of the vein by the subjective soreness. Deep pelvic soreness was complained of in two cases, and was approximately localized to the vicinity of the iliac veins.

Subjective Symptoms.—The usual complaint was a localized, deep soreness, present during rest and often described as a deep, burning, dull ache, generally aggravated by moving the limb and by pressing on the tissues along the affected vessel.

Edema.—Demonstrable edema, graded 1 or 2, was present in 50 per cent of the cases. A careful search was made for swelling over the malleolar and gluteal areas and the shin bone. The absence of edema in half the cases would suggest that the thrombosis was limited to the more

superficial vessels, and to the short and long saphenous veins. The presence of adequate collateral venous circulation prevented fluid stasis. Seventeen per cent of the patients who had been afflicted with edema had a slight residual edema at the time of their dismissal. The massive brawny form of edema and phlegmasia alba dolens were not observed.

Systemic Reactions.—Fever was present in some degree in 89 per cent of the cases. The mean temperature was 100 F. (± 0.8), the highest was 104 F. In ten cases there was no fever. The average duration of fever was three and one-half days, ranging from one to ten days in the uncomplicated cases. The mean fever was determined in the cases of phlebitis without pulmonary infarction and in those with infarction; in the former group the mean maximal temperature was 100.5 F. (± 1); in the latter, the mean temperature was 100.6 F. (± 1.4). The probable error of these mean temperatures eliminated any difference between the fever in the two groups. In the group with pulmonary infarction, the range of duration was from one day to three weeks. The pulse rate was not greater than 100 in the majority of cases. The leukocyte response was slight, the count averaging 11,000 for the group, which is about the normal postoperative value. The highest count was 19,500 for each cubic millimeter. The average leukocyte count in the uncomplicated cases of phlebitis was 9,000 for each cubic millimeter; that in cases with pulmonary infarction was 13,500 for each cubic millimeter.

Pulmonary Complication.—Pulmonary infarction was recognized clinically in twenty-nine cases (33 per cent). The signs and symptoms of the condition were usually typical, consisting of pleuritic pain, slight hemoptysis with a demonstrable area of impaired resonance and friction rub. The signs and symptoms of infarction appeared on an average of four days after the onset of phlebitis, but in three cases they preceded the signs of phlebitis. The mean maximal rise in temperature in this group was 100.6 F. (± 1.4), and the average duration of fever was five days, ranging from one to twenty-one days. In no instance was there sufficient pleurisy with effusion to require paracentesis. The results of roentgenologic examination were frequently negative, or "an area of congestion or bronchopneumonia" was reported. Pulmonary embolism was not a complication, nor was serious disability noted as the result of the pulmonary infarction. The usual clinical diagnosis was bronchopneumonia from pulmonary infarction.

DEGREE OF DISABILITY

The degree of disability as a result of phlebitis was slight among the eighty-seven cases. In only two cases was edema present after six months.

ETIOLOGY

Evidence pointing to the direct cause of the phlebitis was not obtained. The signs and symptoms indicated an infectious lesion of the wall of the vein and secondary thrombosis. A study of resected segments of veins in superficial types of phlebitis revealed marked inflammatory

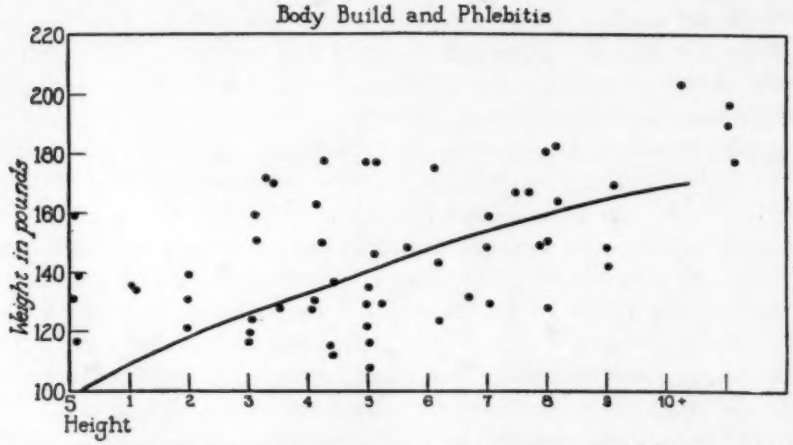

Fig. 2.—Cases of phlebitis charted according to weight and height; distribution rather even for all builds.

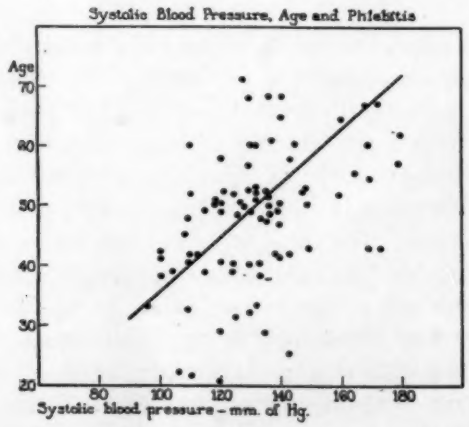

Fig. 3.—Incidence of phlebitis with regard to the systolic blood pressure at various ages. The majority of cases are within the normal range of pressure for the age.

reaction in all coats of the vein and the contiguous tissue. The vessel was markedly infiltrated with polymorphonuclear leukocytes. The obturating thrombus was composed of erythrocytes, leukocytes and fibrin. The reaction was intense and suggested an inflammatory process on an infectious basis.

Possible Predisposing Factors.—Sixty-six per cent of the patients in the series were women and 34 per cent were men. The ages varied from 18 to 72 years. The mean age for the group was 47.1 (± 0.8) years. The mean weight was 151.6 (± 1.7) pounds, and 15 per cent of the patients were definitely obese. There was no demonstrable correlation between phlebitis and body build in the curves on weight and height (fig. 2). The factors of age and systolic blood pressure were considered in relation to phlebitis (fig. 3). The mean systolic pressure was 131.5 (± 1.3). Correction was not made in the systolic blood pressure for age, weight and sex. It will be seen that while the greatest incidence was between the fifth and sixth decades, the majority of cases fall within the range of blood pressure that was normal for the age.

Types of Preceding Operation.—Phlebitis followed operations of the intra-abdominal type in 84.5 per cent of the cases; fifteen and one-half

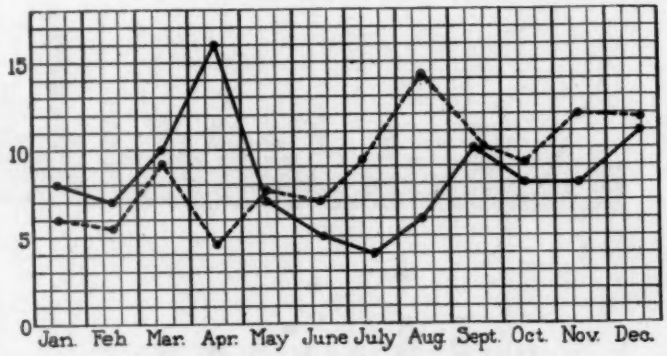

Fig. 4.—Monthly percentage incidence of cases of phlebitis, from 1923 to 1925, inclusive, contrasted with monthly incidence of fatal cases of pulmonary embolism. The solid line indicates cases of phlebitis; the broken line, cases of embolism.

per cent were extraperitoneal; 40 per cent of the abdominal operations were performed on the pelvis and 60 per cent on the stomach, gallbladder and appendix. There was one instance of phlebitis following removal of the breast.

SEASONAL INCIDENCE

There has been a clinical impression that phlebitis has a seasonal incidence. Figure 4 shows the monthly incidence of the cases from 1923 to 1925 inclusive, with the monthly incidence of fatal cases of pulmonary embolism for the same years. The incidence curves are not comparable. Figure 5 gives a more detailed graphic analysis. The number of surgical entrants to St. Mary's Hospital is shown for each month from 1923 to 1925. The number of cases of fatal postoperative

embolism and postoperative phlebitis are shown for the same periods. It will be observed that in about half the months there is a rough correlation between the greatest number of surgical entrants and the highest incidence of pulmonary embolism. Such a correlation apparently does not exist between the surgical entrants and the cases of phlebitis. Figure 6 shows curves for the total number of cases of embolism and phlebitis and surgical entrants for the three year period. The curves for the cases of embolism and surgical entrants show some relationship,

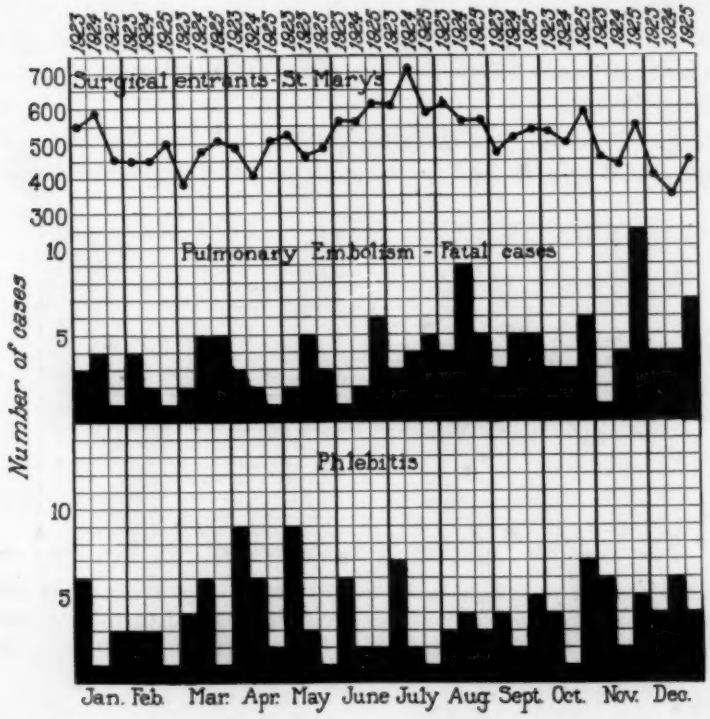

Fig. 5.—Comparison of surgical entrants with incidence of pulmonary embolism and of phlebitis by monthly periods for three years.

while the curve for the cases of phlebitis seems to be independent of the curve of the surgical entrants. There is a spring and fall incidence producing two peaks in the curve for phlebitis which is different from the curve for postoperative embolism.

RELATIONSHIP OF POSTOPERATIVE PHLEBITIS TO EMBOLISM

Henderson showed in his study on postoperative fatal pulmonary embolism that probable phlebitis was present in twenty-nine cases (11 per cent). This incidence of phlebitis is probably higher than the actual

incidence, as the minimal standards for its diagnosis was not carried out in many of his cases. In my series of eighty-seven cases of phlebitis, fatal pulmonary embolism did not occur once, while pulmonary infarction was recognized in 33 per cent. It is probable that if the chest were carefully examined in every case of phlebitis, regardless of whether the chest produced symptoms, the incidence of pulmonary infarction would be higher.

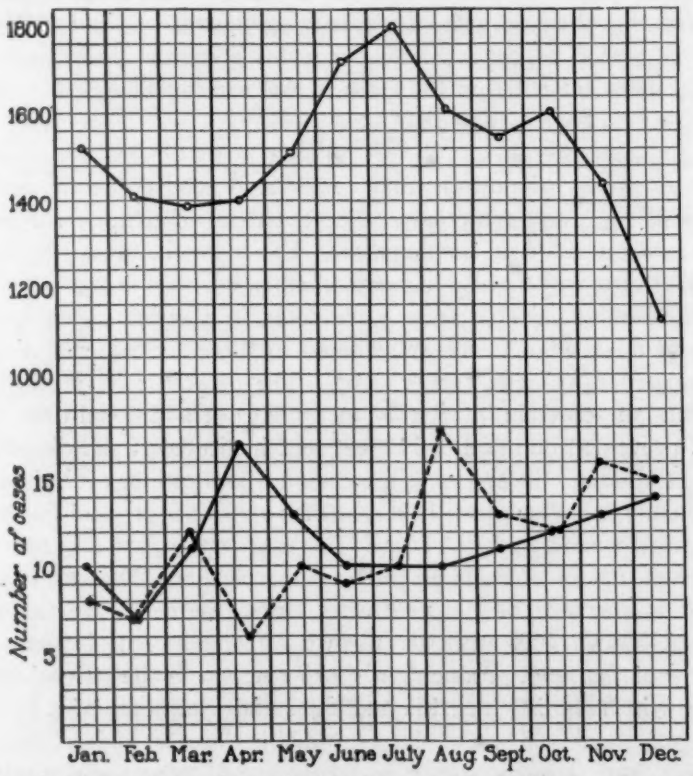

Fig. 6.—Comparison of surgical entrants with incidence of postoperative phlebitis and of postoperative embolism during the years 1923, 1924 and 1925; monthly average for three years. The solid line indicates cases of phlebitis; the broken line, cases of embolism.

TREATMENT AND PROGNOSIS

The treatment of phlebitis has consisted of elevation of the involved limb, heat applied locally and enforced rest. The average length of time for treatment was fourteen days. In 17 per cent of the cases there was some residual edema at the time of the patient's dismissal. These patients were advised to carry out postural exercises, as for obliterative arterial disease. They consist in elevation of the leg to 180 degrees for one minute, lowering it for one minute, then letting it rest in a horizontal

position for two minutes. These exercises are repeated ten times, and the series is repeated two or three times daily until the postural edema has disappeared. If these measures do not suffice, bandaging is advised. The cloth elastic bandages are applied from the foot to the middle of the thigh in the morning before the patient arises, and they are changed at least three times daily. In only two cases did signs of edema persist longer than six months.

The treatment of pulmonary infarction consists in strapping the chest for the pleuritic pain, administering opiates and prescribing an additional period of rest. Serious sequelae from the pulmonary complication were not noted in the cases in this series.

COMMENT

The diagnosis of postoperative phlebitis is too frequently made on insufficient signs and symptoms. The existence of pain or soreness in the leg is the usual basis for diagnosis. Exact localization of the tender or painful area will usually indicate whether the vein is the seat of the trouble. Tender palpable areas were noted in a series of eighty-seven cases, and these were fairly well limited in three. Frequently the vein was palpated as a firm, tender cord. The diagnosis of phlebitis has more than an academic interest, as many days of additional time in bed may be prevented or more rigid treatment instituted if the diagnosis is certain. Pulmonary infarction is a common complication, while fatal pulmonary embolism is apparently rare. This verifies a surgical impression of the relative safety of phlebitis. The explanation for this must rest on the fact that phlebitis is an inflammatory lesion. The clot is firmly attached to the wall of the vein, and large fragments are not easily dislodged. Small fragments are thrown off and become lodged in the periphery of the lung, producing a sharp reaction in the parenchyma of the lung with resulting pleuritis and signs and symptoms of a localized bronchopneumonia. The dislodged thrombi in the cases of fatal embolism are larger, and frequently long segments are dislodged and carried to the lung. Their attachment to the wall of the vein is insecure, and dislodgment is easy. Further evidence on the probable essential difference in the nature and behavior of the cases of phlebitis and fatal embolism is shown by the seasonal incidence. The incidence curve of fatal pulmonary embolism seems to follow roughly the curve of the surgical entrants. Phlebitis suggests a seasonal incidence similar to that observed in duodenal ulcer. The spring and fall colds and infections of the upper respiratory tracts may be factors. While the pathologic appearance and the clinical course of phlebitis suggest an infectious origin, the bacteriologic agent is not proved. In a series of cases of idiopathic superficial phlebitis, culture of a portion of the inflamed vessel and contained clot have resulted negatively. No correlation has been determined to

exist between phlebitis and the factors of age, weight and systolic blood pressure, factors which seem to play a contributory rôle in cases of embolism, as shown by Henderson and Snell.

SUMMARY

In a series of eighty-seven carefully studied cases of postoperative phlebitis, the most characteristic diagnostic sign was a localized tenderness of the affected vein. The presence of edema is equivocal. Pulmonary infarction was a frequent complication, but no instance of fatal pulmonary embolism was encountered. Comparison of the seasonal incidence and predisposing factors in phlebitis and pulmonary embolism shows certain clear-cut differences. The degree of disability due to postoperative phlebitis was notably slight in this series of cases.

CHANGES IN THE BLOOD FOLLOWING OPERATION

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The relatively high incidence of vascular thrombosis leading to pulmonary embolism in surgical patients as compared with medical patients is well known, but the cause is still obscure. The implication is that the surgical procedures, per se, or their sequelae, are responsible for the occurrence of pulmonary embolism. Since primarily the embolus is a clot dislodged from some distant locus, attention is directed toward the mechanism of blood clotting and the factors that might

Cases Studied for Changes in the Blood Following Operation

Case	Age, Sex*	Height, Inches	Weight, Pounds	Operation	Post-operative Complications	Length of Operation, Minutes	Anesthetic
1	65, ♂	71	165	Andrews Bassini herniotomy..	None	28	Regional
2	50, ♂	70	136	Posterior gastro-enterostomy	None	45	Ethylene, oxygen and ether
3	47, ♂	72	188	Posterior gastro-enterostomy	None	50	Ethylene, oxygen and ether
4	46, ♀	65	158	Cholecystectomy.....	None	50	Ethylene, oxygen and ether
5	43, ♀	—	195	Total abdominal hysterectomy	None	47	Ethylene, oxygen and ether
6	48, ♀	65	169	Cholecystectomy.....	Phlebitis	50	Ether
7	45, ♂	70	175	Andrews Bassini herniotomy..	None	60	Ether
8	48, ♂	67	178	Bilateral Andrews Bassini herniotomy	None	45	Ether
9	31, ♀	62	168	Posterior gastro-enterostomy	None	55	Ether
10	62, ♂	60	160	Cholecystectomy, posterior gastro-enterostomy	None	81	Ether
11	55, ♀	60	118	Cholecystectomy, appendectomy	None	40	Ether
12	44, ♀	66	160	Radical amputation of the breast	None	55	Ether

* In this table, ♂ indicates male; ♀, female.

participate in the formation of a clot within a vein following operation. In order to determine what effect, if any, a major operation might have on the condition of the blood, twelve patients were observed for changes in the blood following operation at the Mayo Clinic (table 1). Five determinations were made of each factor studied. The first determination was made within two hours before operation, the second within four hours after operation, the third on the third day following operation, the fourth on the sixth day following operation and the fifth on the tenth day following operation.

OBSERVATIONS ON PATIENTS AFTER EXAMINATION

Erythrocytes.—Erythrocytes were counted from capillary blood. With two exceptions, the decrease in erythrocytes occurred immediately after operation. In cases 3 and 7 the decrease occurred between the second and third readings. The decrease in the number varied between

200,000 in case 7 and 1,000,000 in case 2. The approximate average decrease for the entire group was 600,000 (12.5 per cent). The average weight of these patients was 75 Kg. The average volume of whole blood was 6,750 cc. (90 cc. for each kilogram of body weight). The amount of blood lost at operation which would cause a diminution of 600,000 erythrocytes then could be calculated as 843 cc. (12.5 per cent of 6,750 cc.). Obviously this loss does not occur in such simple operations as unilateral herniotomy. The observations on an additional group of fourteen patients show that the decrease in the number of erythrocytes immediately after operation is not as constant as is indi-

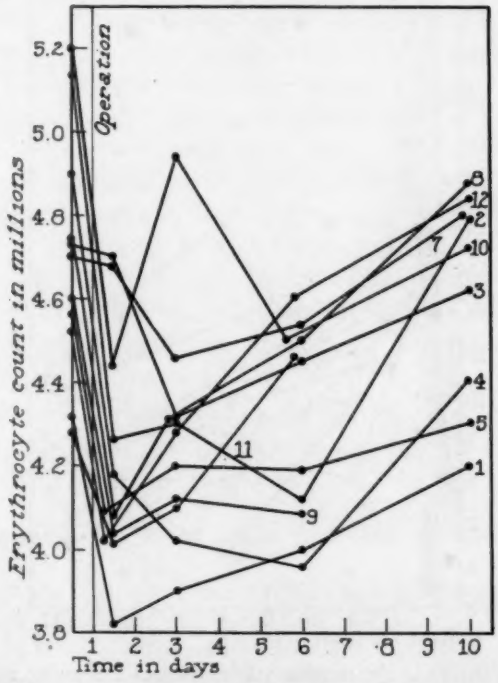

Chart 1.—Change in the number of erythrocytes following operation.

cated in chart 1. In some instances there was an immediate postoperative increase of 1,000,000 cells. The number of erythrocytes in oxalated blood drawn from the median basilic vein and from the capillary blood were practically identical. The increase in the erythrocytes was gradual, usually beginning at the second reading and being complete at the fifth. Many factors influence the number of erythrocytes, as has been shown by Bostrom,[1] Boycott and Jones,[2] Schneider

1. Bostrom, E. F.: Conditions Causing an Unequal Distribution of Erythrocytes in the Blood-Stream, Am. J. Physiol. **58**:195, 1921.

2. Boycott, A. E., and Jones, C. P.: The Influence of Anesthesia on the Restoration of the Volume of the Blood After Haemorrhage and After Transfusion, Jour. Path. & Bact. **25**:335, 1922.

and Havens,[3] Scott and his co-workers,[4] and von Lesser.[5] These investigators noted that the number of the erythrocytes was influenced by vasoconstriction, exercise, abdominal massage, abdominal pressure, changes in blood pressure, the anesthetic and the amount of blood lost at operation. Their observations and this study indicate that many factors influence the number of erythrocytes following operation, among which peripheral vasomotor changes, changes in the blood pressure, rest in bed, trauma within the abdomen and abdominal bandaging are the most important.

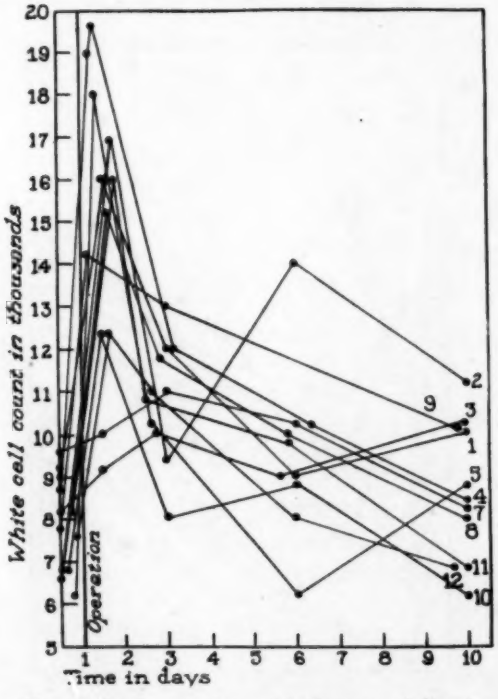

Chart 2.—Change in the number of leukocytes following operation.

Leukocytes.—Leukocytes were counted in the capillary blood. A marked increase was noted in the number of leukocytes for each cubic millimeter of blood at the second reading, except in cases 3 and 7 (chart 2). This increase varied from 11,500 cells in case 4 to less than

3. Schneider and Havens, quoted by Bostrom (footnote 1).

4. Scott, F. H.: Factors Influencing the Interchange of Fluid Between Blood and Tissue Spaces. I. Blood Pressure, Am. J. Physiol. **44**:298, 1917. Scott, F. H.; Herrmann, E. T., and Snell, A. M.: Factors Influencing the Interchange of Fluid Between Blood and Tissue Spaces. II. Muscular Activity, Am. J. Physiol. **44**:313, 1917.

5. Von Lesser, quoted by Scott (footnote 4).

5,000 in case 12; the average increase was approximately 8,000 cells. The decline in the number of leukocytes was almost as abrupt as the increase, being marked between the second and third readings and more gradual between the third and fifth. An unexplained increase in the number of leukocytes occurred between the third and fourth readings in case 2. In most cases, return to the preoperative level was not complete until the fifth reading. That the leukocytes do not increase in the capillary blood alone is shown by data from fifteen additional patients from whom the blood was removed simultaneously from the ear and from the median basilic vein. The average postoperative increase of leukocytes in the venous blood was 12,500 and in the capillary blood 13,900. The number of leukocytes in the blood from the ear average 2,500 cells higher than in the venous blood. Discrepancies in the number of leukocytes in the capillary and venous blood have been studied by Yarbrough[6] and Foord.[7] The factors that influence the number of leukocytes have been studied by Shaw,[8] Mora, Amtman and Hoffman,[9] Isaacs and Gordon,[10] and Dawson.[11] From their observations and from the evidence derived from the study of this series of cases, it is probable that the consistent increase in the number of leukocytes following operation is due to many factors, the more important of which are the release of leukocytes from temporary "storage areas," peripheral vasoconstriction, the mental status of the patient and trauma to tissues.

Fibrin.—Estimations of fibrin were made by the method of Foster and Whipple.[12] There was a definite increase in two cases and a decrease in one case at the second reading. In all cases there was a

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6. Yarbrough, Nancy: Blood Counts with Oxalated Blood, J. Lab. & Clin. Med. **7**:172, 1921.

7. Foord, A. G.: Blood Counts With Oxalated Blood Compared With Ordinary Counts, J. Lab. & Clin. Med. **8**:343, 1922.

8. Shaw, A. F. B.: Influence of Vasomotor State of Peripheral Blood-Vessels on Leukocytic Content of Blood, J. Path. & Bact. **29**:389, 1926.

9. Mora, J. M.; Amtman, L. E., and Hoffman, S. J.: Effect of Mental and Emotional States on the Leukocyte Count, J. A. M. A. **86**:945 (March 27) 1926.

10. Isaacs, Raphael, and Gordon, Burgess: The Effect of Exercise on the Distribution of Corpuscles in the Blood Stream, Am. J. Physiol. **71**:106, 1924.

11. Dawson, F. I.: The Condition of the Blood After Operation and Fracture, Edinburgh M. J. **18**:426, 1905.

12. Foster, D. P., and Whipple, G. H.: Blood Fibrin Studies: I. An Accurate Method for the Quantitative Analysis of Blood Fibrin in Small Amounts of Blood, Am. J. Physiol. **58**:365, 1922; II. Normal Fibrin Values and the Influence of Diet, ibid, p. 379; III. Fibrin Values Influenced by Transfusion, Hemorrhage, Plasma Depletion and Blood Pressure Changes, ibid, p. 393; IV. Fibrin Values Influenced by Cell Injury, Inflammation, Intoxication, Liver Injury and the Eck Fistula, ibid, p. 407.

marked increase at the third reading (chart 3); this averaged approximately 175 mg. for each hundred cubic centimeters of blood (41 per cent increase over the original average reading). The variation is more marked than that which occurs normally.[12] The fibrin content between the third and fifth readings varied, increasing or decreasing slightly. A marked increase was noted between the fourth and fifth readings in two cases. The values were uniformly high on the tenth day, and there was no indication of a return to the preoperative level.

After an intensive study of the effect of many factors, Foster and Whipple concluded that trauma to the tissues is the most powerful single stimulus to the overproduction of fibrin in the blood. Schultz, Nicholes and Schaefer [13] and Smotrov [14] studied the influence of additional factors on the amount of fibrin. Smotrov found high values in patients with syphilis, cancer and other conditions, and was convinced that the

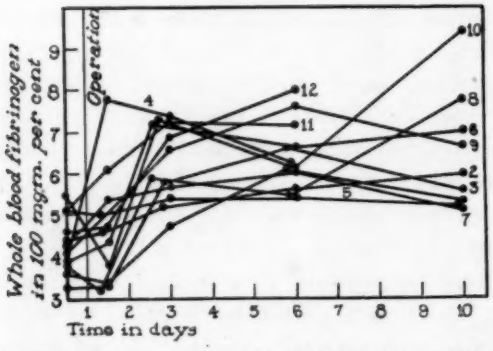

Chart 3.—Changes in the amount of fibrin in the blood following operation.

increase in fibrin is not due to the withdrawal from the liver but to a decomposition of albumin in many tissues. This has not been the opinion of Foster and Whipple. Smotrov could not demonstrate any relationship between the amount of fibrinogen and the time of coagulation of the blood, although the size and resistance of the clot seemed to parallel the amount of fibrin in the blood. In view of the foregoing and the absence of wound infections, it would appear that the almost doubled amount of fibrin in the cases of this series was probably due to trauma, which may have produced a demand for an increase in fibrin at the site of the wound.

Bleeding Time.—There were no uniform variations in the bleeding time. The divergent results probably indicate errors in calculation.

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13. Schultz, E. W.; Nicholes, J. K., and Schaefer, J. H.: Studies on Blood Fibrin. Its Quantitative Determination; Normal Fibrin Values, and Factors Which Influence the Quantity of Blood Fibrin, Am. J. Path. **1**:101, 1925.

14. Smotrov, V. N.: Russk. klin. Mosk. **3**:843, 1925.

Platelets.—There were no uniform variations in the number of platelets. With the exception of case 6, in which the count was low at all readings, and that of case 7, in which it was uniformly high, the number of platelets was approximately normal, varying from 200,000 to 260,000.

Coagulation Time.—Two methods for the study of the coagulation time were used; the older method of Lee and the more recent capillary-tube method of Petersen and Mills.[15] The results with the Lee method were variable and seemed of little significance. With the Mills method, there was an increase in coagulation time between the first and second readings in five cases and a decrease in three cases; there was a decrease between the third and fourth readings in all cases, except in cases 5 and 12. The results were widely divergent between the fourth and fifth readings (chart 4). Smotrov[14] and Nizner[16] did not find any consistent

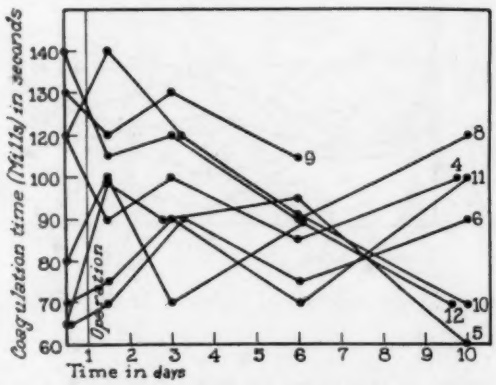

Chart 4.—Change in coagulation time following operation.

changes in the coagulability of the blood following operation. Nizner believed that the time of coagulation of the blood is variable, depending on such factors as hunger, catharsis, enemas and the patient's mental and physical state. He concluded that patients in whom the coagulation time of the blood is increased following operation are more liable to postoperative complications. This observation could not be confirmed in the small group of cases presented here. Straus and Rubin[17] found a transitory but definite increase in bleeding and coagulation time following ethylene anesthesia. The changes in the coagulation time in this series of cases were slight and inconstant.

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15. Petersen, M. F., and Mills, C. A.: A New Method for Accurately Determining the Clotting Time of the Blood, Arch. Int. Med. **32**:188 (Aug.) 1923.

16. Nizner, E.: Russk. klin. Mosk. **1**:522, 1924.

17. Straus, D. C., and Rubin, H. H.: The Coagulation Time in Ethylene Anesthesia, J. A. M. A. **88**:310 (Jan. 29) 1927.

Blood Calcium.—With the exception of cases 7, 8 and 9, there was a decrease in the amount of calcium in the blood at the second reading, varying from 0.4 to 1.0 mg. for each hundred cubic centimeters (chart 5). A further decrease was noted in several cases at the third reading. In the remaining cases there was approximately the same amount of calcium at the third reading as at the second, except in cases 11 and 12, which showed increased amounts. There were no constant variations in the amount of calcium in the blood following the third reading. Sakaian found that narcosis alone did not produce any change in the calcium content of the blood. He also showed that great losses of blood during operation in some cases produced an increase in the amount of calcium and in others a decrease, associated with an increase or decrease in the coagulability of the blood. This relationship could

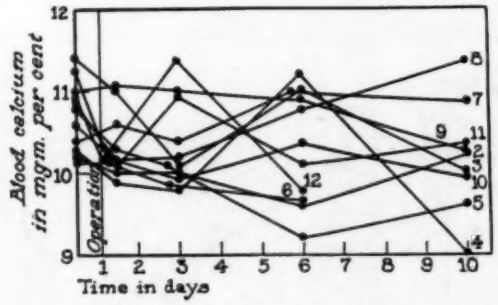

Chart 5.—Change in the amount of calcium in the blood stream following operation.

not be demonstrated in the cases of this series, and the changes in the amount of calcium in the blood did not seem great or constant enough to be of significance.

Prothrombin Time.—The prothrombin time was calculated by the method of Howell.[18] The results in one case are shown in chart 6. The numbers on the curves indicate the tube number in the series. Each curve (for example, the first curve in chart 4) represents the variation in the prothrombin time on several days in a tube containing constant ingredients, the only known possible variation being in the quality (not quantity) of the blood introduced at successive tests. The results in the other nine cases are similar to those shown in chart 4; there was a sharp increase in the prothrombin time at some time following operation. In all cases the sharp increase was followed by an equally sharp decrease. If an increase in the prothrombin time indicates a decrease in the amount

18. Howell, W. H.: The Condition of the Blood in Hemophilia, Thrombosis and Purpura, Arch. Int. Med. **13**:76 (Jan.) 1914.

of prothrombin in the blood following operation, it might be explained on the basis of the utilization of prothrombin in the transformation of fibrinogen into fibrin.

Lipoids and Cholesterol.—Fatty acids were calculated by the method of Bloor, Pelkan and Allen.[19] In cases 1, 2, 3 and 7 no significant changes were observed. In cases 5, 6 and 13 the readings were originally high but dropped rapidly between the first and fourth readings, and none reached the preoperative level by the tenth day (chart 7). The results in these three cases are of interest largely because all the patients were obese.

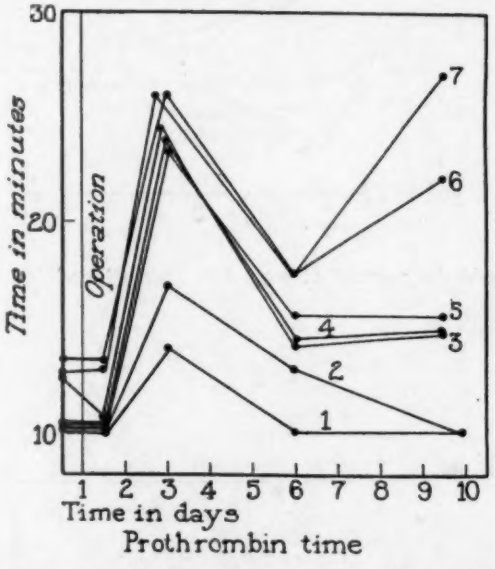

Chart 6.—Change in prothrombin time in one patient following operation.

Lecithin was determined by the method of Whitehorn.[20] A sharp diminution in the amount of lecithin occurred in all cases (chart 8); in some cases between the second and third readings but more constantly between the third and fourth readings. Results between the fourth and fifth readings were variable.

The total fats were calculated by the addition of the values for the fatty acids and the lecithin. There was a uniform decrease in the amount between the first and fourth readings (chart 9); it remained unchanged or increased slightly following the fourth reading, except in case 3.

19. Bloor, W. R.; Pelkan, K. F., and Allen, D. M.: Determination of Fatty Acids (and Cholesterol) in Small Amounts of Blood Plasma, J. Biol. Chem. **52**:191, 1922.

20. Whitehorn, J. C.: A Method for the Determination of Lipoid Phosphorus in Blood and Plasma, J. Biol. Chem. **62**:133, 1924.

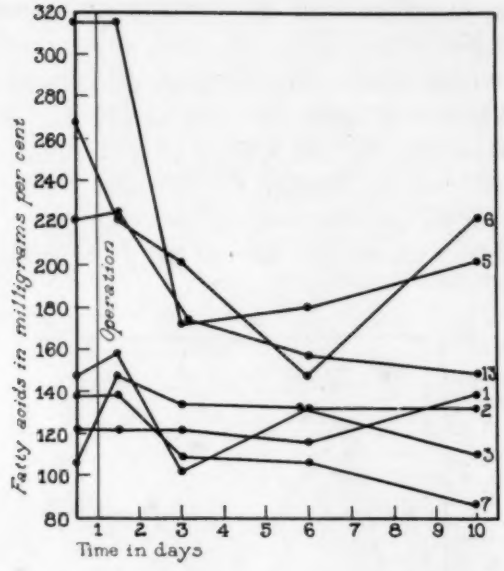

Chart 7.—Change in the amount of total fats in the blood following operation.

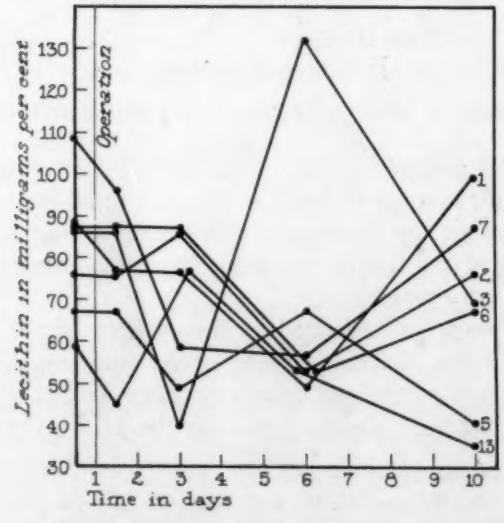

Chart 8.—Change in the amount of lecithin following operation.

The cholesterol content of the blood was studied by the Bloor [21] method. There were no constant variations which could be considered significant.

Atkinson and Ets [22] found that 1 grain (0.06 Gm.) of morphine sulphate given to dogs caused a decrease of 13 per cent of cholesterol and 9 per cent of lecithin, but increased the total fat 10 per cent. Bloor [23] could show no effect of morphine on the amount of total fat. In this series of cases, high fatty acid values in obese patients were sharply diminished following operation, the amounts of lecithin and total fats were definitely diminished and the cholesterol values were unchanged. Morphine narcosis alone probably could not account for these changes.

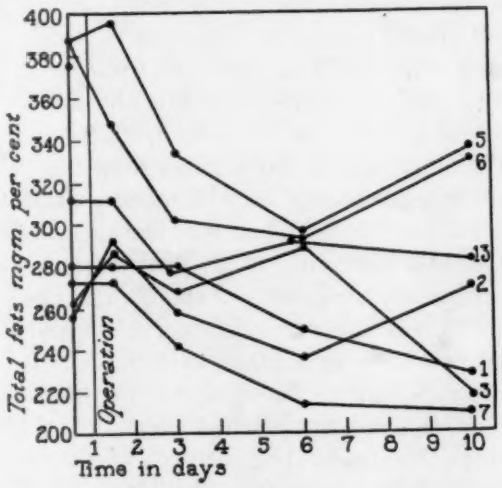

Chart 9.—Changes in the amounts of total fats in the blood following operation.

COMMENT

The number of platelets, the cholesterol, the bleeding time and the coagulation time (Lee) do not show definite change and do not require comment. The coagulation time (Mills) and the blood calcium show slight variations but not consistently enough to be considered of significance. The number of erythrocytes and leukocytes, the prothrombin time, the fibrinogen and the lipoids show definite and constant changes

21. Bloor, W. R., and Knudson, Arthur: The Separate Determination of Cholesterol and Cholesterol Esters in Small Amounts of Blood, J. Biol. Chem. **27**:107, 1916. Bloor, W. R.: Studies on Blood Fat. II. Fat Absorption and the Blood Lipoids, J. Biol. Chem. **23**:317, 1915.

22. Atkinson, H. V., and Ets, H. N.: Chemical Changes of the Blood Under the Influence of Drugs: II. Morphine, J. Lab. & Clin. Med. **8**:170, 1922.

23. Bloor, W. R.: Studies on Blood Fat. I. Variations in the Fat Content of the Blood Under Approximately Normal Conditions, J. Biol. Chem. **19**:1 1914.

which are in sharp contrast to the other factors studied. Walters and Hendricks [24] have demonstrated similar variations in the fibrin, calcium, leukocytes and coagulation time. The fibrinogen is probably the most important of these changes. Essential as it is in the process of blood coagulation, the sharp constant increase following operation is doubtless significant in intravascular coagulation. The action of the fibrinogen may be manifested by the increased size and strength of the clot (Smotrov), although there is no apparent parallel change in the coagulation time. The postoperative increase in the number of leukocytes is significant because leukocytes are known to furnish thromboplastic substances that play an important part in the coagulation of the blood. The sharp prolongation of the prothrombin time is important, although the interpretation is obscure. The change in the number of erythrocytes may indicate widespread vasomotor disturbances, which definitely influence intravascular clotting. The consistent decrease in the amount of the phospholipoid, lecithin and the total fats and the diminution of the fatty acids in obese patients are of interest, although their importance is not clear. The other papers in this series show the influence of many other factors on the occurrence of fatal pulmonary embolism. Studies by Johnson [25] and Brown [26] have shown that anesthesia and inflammatory postoperative phlebitis have little or no demonstrable influence on the occurrence of pulmonary embolism. Although this study is based on a small number of clinical and surgical types, it is apparent that changes in the blood were consistent in response to operation. It is probable that with the possible exception of the lipoids in the blood, there is a nonspecific physiologic response to operation which occurs independently of the clinical or surgical status of the patient. This nonspecific physiologic change produces definite changes in the blood which may partially explain the relatively high incidence of pulmonary embolism occurring in surgical patients as compared with nonsurgical patients. There is probably in every surgical patient a definitely increased potentiality for intravascular coagulation by virtue of these changes, but this in all probability plays a minor part in comparison with other factors in the actual deposition of the clot and the subsequent pulmonary embolism.

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24. Walters, Waltman; and Hendricks, W. A.: Unpublished data.

25. Johnson, A. C.: Unpublished data.

26. Brown, G. E.: Postoperative Phlebitis, A Clinical Study, Arch. Surg. published in this issue.

PLASMA PROTEINS IN CUTANEOUS BURNS*

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AND
C. W. MATTHEW
DETROIT

Attention has frequently been called to the analogy between traumatic toxemia and the toxemia following extensive burns. Certain alterations in the plasma proteins have been observed which are of fundamental importance in explaining the syndrome in shock. It, therefore, seemed desirable to study the plasma proteins of a group of cases of burns to determine whether further light might be thrown on the toxemia which is seen after recovery from the initial collapse in such cases.

Starling [1] demonstrated that the walls of the blood vessels are normally impermeable to colloids. It is also known [2] that the composition of the plasma of a given person is relatively constant. Dale and Laidlaw [3] have shown, however, that the concentration of blood in histamine shock is not accompanied by an increase of the plasma proteins. They concluded that histamine acted directly on the capillary endothelium, rendering it more permeable, and that whole plasma rather than simply water escaped into the tissues. Bodansky [4] subsequently studied the plasma proteins in experimental anhydremia produced by histamine and reached the same conclusion. Gasser, Erlanger and Meek [5] likewise observed filtration of whole plasma into the tissues in experimental traumatic shock.

Bayliss [6] states that, since it is the osmotic pressure of the proteins that prevents the rapid filtering away of the liquid portion of the blood into the tissue spaces and causes reabsorption of fluid from them, it can be effective only as long as the concentration of the proteins is unequal on the two sides of the membrane. It is clear that if the proteins are

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1. Starling, E. H.: On the Absorption of Fluids from the Connective Tissue Spaces, J. Physiol. **19**:312, 1895-1896.

2. Howe, P. E.: The Function of the Plasma Proteins, Physiol. Rev. **5**:439, 1925.

3. Dale, H. H., and Laidlaw, P. P.: Histamine Shock, J. Physiol. **52**:355, 1918-1919.

4. Bodansky, M.: Plasma Proteins in Experimental Anhydremia, Proc. Soc. Biol. Chemists, twentieth meeting, J. Biol. Chem. **67**:38, 1926.

5. Gasser, H. S.; Erlanger, J., and Meek, W. J.: Studies in Secondary Traumatic Shock. IV. The Blood Volume Changes and the Effect of Gum Acacia on Their Development, Am. J. Physiol. **50**:31, 1919-1920.

6. Bayliss, W. M.: The Action of Gum Acacia on the Circulation, J. Pharmacol. & Exper. Therap. **15**:29, 1920-1921.

diffused through the capillary walls in shock, the concentration will become equal on both sides, and there is no longer any force resisting filtration or causing absorption.

The toxemia of burns is particularly adaptable to the study of capillary permeability, because the symptoms persist for a number of days after the initial anhydremia has been replaced by a more normal fluid balance. This complicating factor has prevented extensive investigation of the problem in traumatic toxemia.

METHODS

The burns were treated by coagulation of the devitalized tissue with tannic acid,[7] and were thereafter kept exposed to air. Fluids were forced energetically in an effort to restore fluid balance promptly.[8] The patients were placed on a constant diet which consisted of approximately 60 Gm. of protein and sufficient fat and carbohydrate to give a total value of about 2,500 calories. Food has little effect on the plasma proteins,[9] but there are transitory changes following the ingestion of water. To insure uniformity, all samples of blood were taken before breakfast. The specimens were collected into test tubes containing powdered potassium oxalate. Generally, 20 cc. of blood were withdrawn, and care was exerted to minimize venous stasis. The whole blood was then centrifugalized for twenty minutes at 2,500 revolutions per minute, and the volume of plasma was determined. The plasma was then pipetted from the cells, and duplicate analyses were made by the method of Wu.[10]

RESULTS

Total Plasma Proteins.—The table gives the results of analyses of blood in ten patients with severe burns. The average total plasma proteins for the group at the expiration of twenty-four hours was 7.03 per cent, while the average volume of plasma was 59.1 per cent (chart 1). These values are considerably lower than those found by Plass and Matthew [11] in normal, nonpregnant women. During the next twenty-four hours the average for the group rose to 7.16 per cent, while the average volume of plasma (chart 2) was slightly lowered to 58.3 per

7. Davidson, E. C.: Tannic Acid in the Treatment of Burns, Surg. Gynec. Obst. **41**:202, 1925.

8. Underhill, F. P.; Carrington, G. L.,; Kapsinow, R., and Pack, G. T.: Blood Concentration Changes in Extensive Superficial Burns and Their Significance for Systemic Treatment, Arch. Int. Med. **32**:31 (July) 1923.

9. Hanson, S., and McQuarrie, I.: The Non-Dependence of the Protein Quotient in the Blood-Serum Upon the Rapidity of Metabolism with Especial Reference to the Non-Effect of Antipyretics, Sodium Cacodylate and Thyroid Extract, J. Pharmacol. & Exper. Therap. **10**:261, 1917.

10. Wu, H.: A New Colorimetric Method for the Determination of Plasma Proteins, J. Biol. Chem. **51**:33, 1922.

11. Plass, E. D., and Matthew, C. W.: Placental Transmission: IV. The Protein Fractions in Fetal and Maternal Plasma, Am. J. Obst. & Gynec. **12**:847, 1926.

Plasma Proteins Following Burns

Case	Date	Plasma Volume, per Cent	Fibrin, per Cent	Albumin, per Cent	Globulin, per Cent	Total Protein, per Cent
1	3/24	50.7	0.25	4.68	....	....
	3/25	46.0	0.46	4.74	2.78	7.98
	3/26	55.3	0.53	4.04	2.14	6.71
	3/27	56.5	0.56	3.90	2.19	6.65
	3/28	55.9	0.73	4.45	2.52	7.70
	3/29	61.9	0.55	4.00	2.34	6.89
	3/30	55.7	0.56	4.51	2.67	7.74
	3/31	58.2	0.52	3.99	2.42	6.93
	4/ 1	53.6	0.50	4.48	2.52	7.50
	4/ 2	57.3	0.36	4.53	2.52	7.41
	4/ 3	54.0	0.47	4.70	2.67	7.84
2	5/17	55.5	0.46	4.42	2.42	7.30
	5/18	59.1	0.55	4.30	2.52	7.37
	5/19	61.2	0.56	4.08	2.09	6.73
	5/20	62.4	0.55	4.00	2.11	6.66
	5/21	62.5	0.50	4.20	2.30	7.00
	5/22	63.5	0.56	3.45	2.44	6.45
	5/23	63.1	0.47	3.97	2.77	7.21
	5/24	61.5	0.45	3.95	2.07	6.47
	5/25	62.5	0.46	3.67	2.39	6.52
	5/26	62.8	0.42	4.17	1.97	6.56
3	5/19	60.2	0.39	4.46	2.55	7.40
	5/20	62.6	0.42	4.20	2.14	6.76
	5/21	61.3	0.44	4.19	2.14	6.77
	5/22	61.3	0.66	4.13	2.75	7.54
	5/23	63.2	0.47	4.64	2.37	7.48
	5/24	62.6	0.41	4.25	2.07	6.73
	5/25	62.4	0.45	4.20	2.24	6.89
	5/26	61.2	0.45	4.96	2.04	7.45
4	6/ 7	67.2	0.41	4.36	2.64	7.41
	6/ 8	63.7	0.44	4.33	2.52	7.29
	6/ 9	68.0	0.45	4.06	2.53	7.01
	6/10	66.7	0.45	4.15	2.53	7.13
	6/11	61.2	0.45	4.23	2.66	7.34
	6/12	57.7	0.40	4.30	2.65	7.35
	6/14	65.6	0.50	4.61	3.12	8.23
	6/15	58.8	0.47	4.35	3.13	7.95
5	8/ 4	66.4	0.35	4.02	2.34	6.71
	8/ 5	61.4	0.50	4.03	3.10	7.63
	8/ 6	57.6	0.47	3.86	3.15	7.48
	8/ 7	58.0	0.49	4.07	3.12	7.68
	8/ 9	60.2	0.46	4.05	3.06	7.57
	8/10	63.2	0.45	4.09	2.96	7.50
	8/11	55.2	0.43	4.00	2.98	7.41
	8/12	59.0	0.45	4.10	2.75	7.30
	8/13	56.4	0.48	4.20	2.70	7.38
6	8/ 5	60.3	0.41	4.71	2.85	7.97
	8/ 6	63.4	0.41	3.99	2.55	6.95
	8/ 7	59.5	0.53	4.02	2.86	7.41
	8/ 9	61.8	0.64	4.31	2.61	7.56
	8/10	62.7	0.73	4.23	2.54	7.53
	8/11	59.1	0.83	4.52	2.17	7.52
	8/13	61.9	0.71	4.32	2.32	7.35
7	9/ 4	40.0	0.57	4.07	2.62	7.23
	9/ 5	55.5	0.66	2.95	2.65	6.26
	9/ 6	62.3	1.10	3.07	2.52	6.69
	9/ 7	63.0	1.25	3.35	2.35	6.95
	9/ 8	61.3	1.46	3.38	2.17	7.01
	9/ 9	59.5	1.38	3.32	2.25	6.95
	9/10	61.4	1.20	3.80	2.10	7.10
	9/11	62.3	0.96	4.00	2.11	7.10
	9/13	63.8	0.86	3.98	2.16	7.00
	9/14	64.0	0.75	4.30	2.20	7.25
8	6/10	63.0	0.26	4.06	1.94	6.26
	6/20	59.0	0.40	4.30	2.10	6.80
9	6/ 5	59.5	0.40	4.20	1.82	6.49
	6/15	60.1	0.41	4.20	2.08	6.69
10	6/10	64.4	0.38	4.50	2.12	7.00
	6/20	65.6	0.39	4.35	2.27	7.01

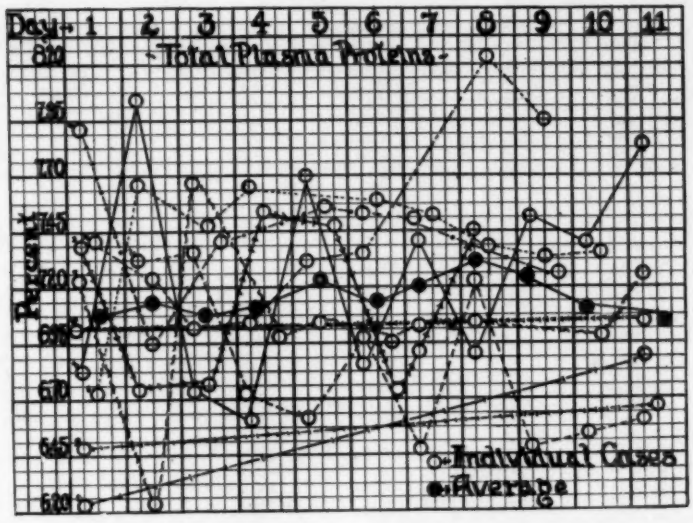

Chart 1.—Total plasma proteins of individual cases and the average curve for the group.

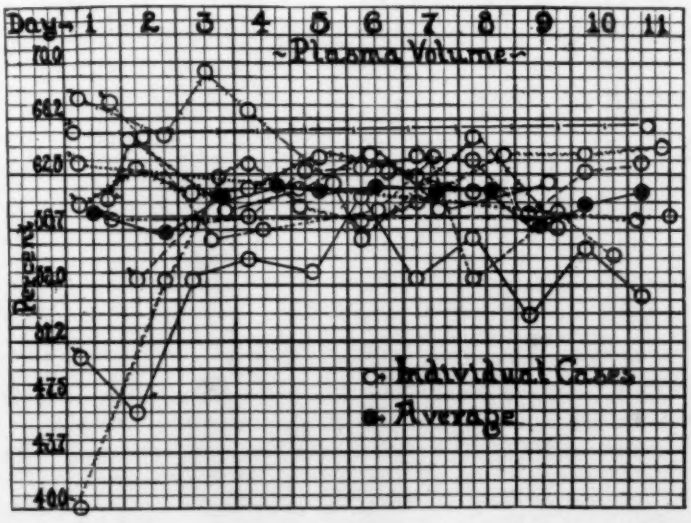

Chart 2.—Percentage volume of plasma for individual cases and the average curve for the group.

cent. This may not present the true sequence of events, because in cases 3, 4, 6 and 7, in which the burns were more serious and extensive than in the others, the plasma proteins showed an appreciable fall after the first twenty-four hours. In case 7, in which there was marked concentration of blood at the end of twenty-four hours, no parallel rise of the total plasma proteins took place (chart 3). As dilution of the blood occurred, there was a prompt fall of the total plasma proteins from 7.26 to 6.26 per cent. The volume of plasma gradually increased to 64.3 per cent on the eleventh day, but during this time the plasma proteins rose to 7.25 per cent, which is essentially the same value as that found on the first day, when the volume of plasma was 40 per cent. The average for

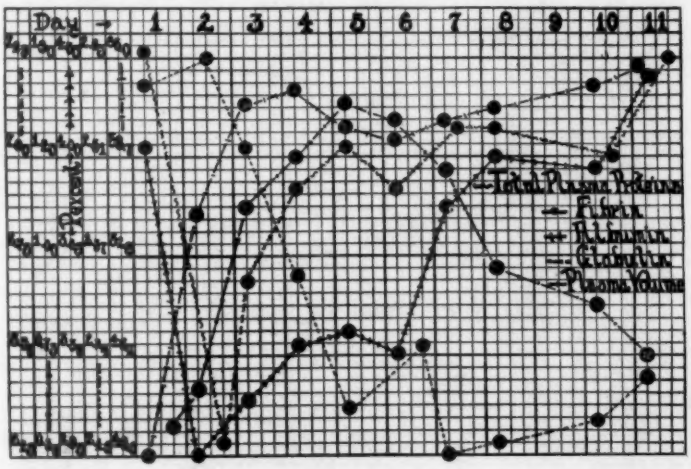

Chart 3.—Curve of the total plasma proteins, fibrin, albumin, globulin, and volume of plasma percentage in case 7.

the series of cases subsequent to the first twenty-four hours showed a gradual rise to 7.39 per cent on the eighth day, and then slowly fell to 7.02 per cent on the eleventh day.

Fibrin.—The results of the fibrin analyses are given in the table. The observations were uniform throughout and showed a gradual rise of the fibrin content of the plasma, which reached its maximum on the fifth day (chart 4), but remained elevated during the period of healing. The average fibrin for the group was 0.35 per cent at the end of the first twenty-four hours. This value is but slightly higher than that given by Foster [12] for normal men. It has been well established that fibrin

12. Foster, D. P.: A Clinical Study of Blood Fibrin; with Observations in Normal Persons, Pregnant Women, and in Pneumonia and Liver Disease, Arch. Int. Med. **34**:301 (Sept.) 1924.

shows the most transitory fluctuations in concentration of any of the plasma proteins. This increased fibrin content of the plasma in patients suffering from burns is similar to that described by Foster and Whipple [13] in a number of conditions in which there was injury and inflammation of tissue.

Globulin.—The table gives the values obtained in analyses for the globulin content. The average for the group at the end of the first twenty-four hours was 2.30 per cent. This amount is somewhat lower than the average 2.69 per cent given by Plass and Matthew for normal, nonpregnant women. A subsequent rise of the globulin content was

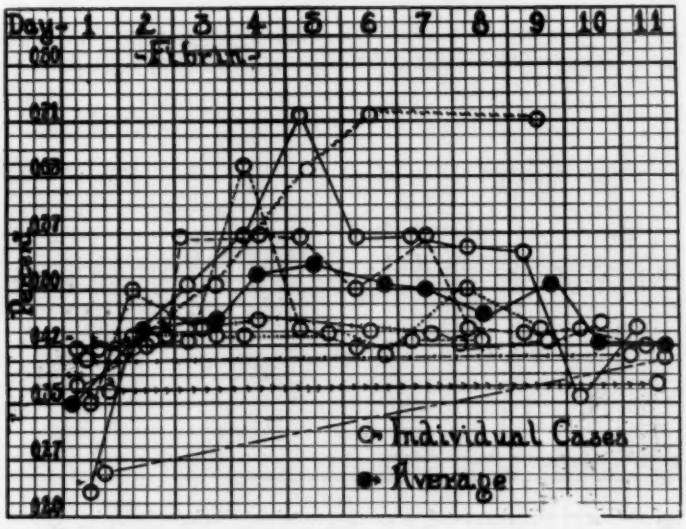

Chart 4.—Fibrin content in individual cases and the a .ge curve for the group. The rise is striking.

noted (chart 5), but this elevation did not reach the normal value. This slight rise is in accord with the observation of Rowe [14] that there is a definite increase in the globulin content of the plasma in most conditions in which infection is present. The fact that the globulin did not change more strikingly under the stimulus of infection and injury may be due to the fact that there was a large initial loss of plasma, and the true extent of the loss was masked by the stimulus to formation of globulin by the burn.

13. Foster, D. P., and Whipple, G. H.: Blood Fibrin Studies. IV. Fibrin Values Influenced by Cell Injury, Inflammation, Intoxication, Liver Injury and the Eck Fistula, Am. J. Physiol. **58**:407, 1921-1922.

14. Rowe, A. H.: The Albumin and Globulin Content of Human Blood Serum, Arch. Int. Med. **18**:455 (Oct.) 1916.

Albumin.—The results of the albumin analyses are shown in the table. The alterations in the albumin content of the plasma in the extensive burns were uniform and were as characteristic as the changes noted for fibrin. The average value at the end of twenty-four hours for the group was 4.34 per cent, which is slightly lower than the average normal value of 4.45 per cent given by Howe.[2] A progressive lowering of the plasma albumin was observed for three days in the curve of the average for the series (chart 6). This was followed by a gradual elevation to normal. In case 7, in which there was the most extensive burn of the series, the depression in the value of the plasma albumin was striking,

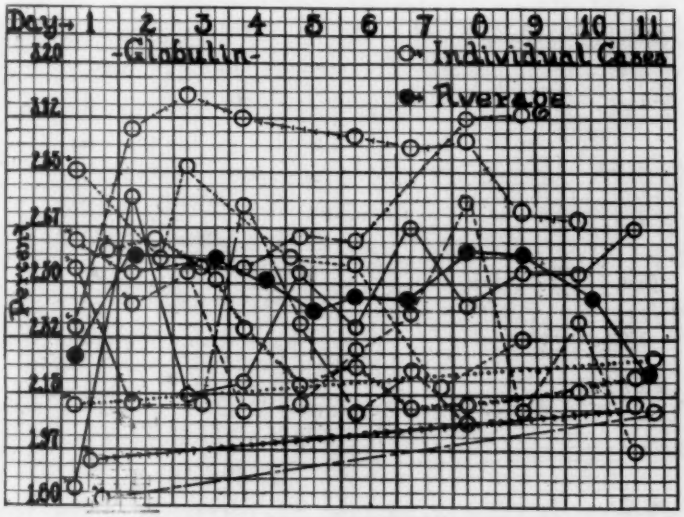

Chart 5.—Globulin content in individual cases and the average curve for the group.

and persisted for ten days. It is conceivable that in an extensive burn there may be a tremendous loss of plasma at the site of the burn, due to the effusion of the inflammatory exudate, and into the tissues in general from increased capillary permeability. Subsequently, when normal fluid balance becomes established dilution of the plasma occurs. A relatively slight difference is found in the total proteins because of the increase in fibrin and globulin content. On the other hand, albumin is regenerated more slowly than either fibrin or globulin,[15] and, accordingly, the analyses show a persistent low value for albumin.

15. Kerr, W. J.; Hurwitz, S. H. and Whipple, G. H.: Regeneration of Blood Serum Proteins. I. Influence of Fasting Upon Curve of Protein Regeneration Following Plasma Depletion, Am. J. Physiol. **47**:356, 1918-1919.

COMMENT

Under normal conditions the composition of the plasma is relatively constant, except for the changes in the fibrin content. According to Howe,[2] the concentration of the proteins varies with age, sex, feeding, physiologic activity, disease and reaction to infection. The changes in endothelial permeability in histamine and traumatic shock indicate possibilities of at least temporary modification in the concentration of proteins through such action.

Age and sex do not need to be considered variable in the present cases, because all of the patients were young men. The effect of diet is known to be of little consequence, but the precaution was taken to

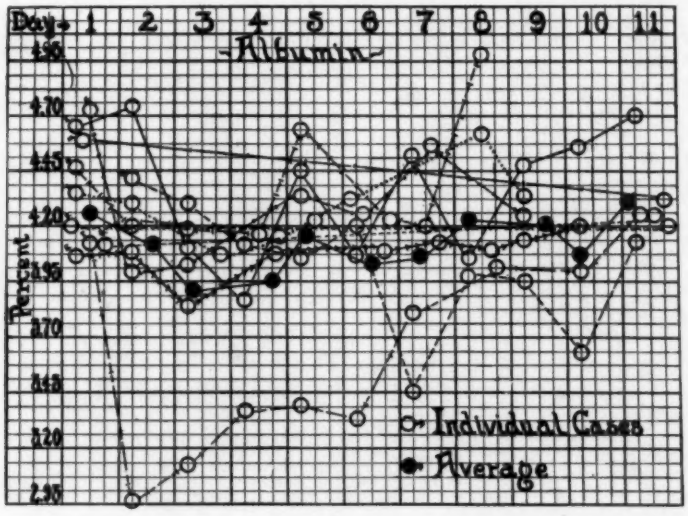

Chart 6.—Albumin content in the individual cases and the average for the group. There is a marked depression of the albumin content.

obtain samples of blood before food was ingested. The activity of the patients was uniform, because the severity of the burns necessitated hospitalization.

The chief variable was the extent and severity of the burn. For instance, in case 7, in which approximately 50 per cent of the total surface of the body was burned, the changes are striking (chart 3), while in the less extensive burns only slight alterations were noted. In this instance, the red blood cell count rose to 7,270,000, and the volume of plasma was 40 per cent eighteen hours after the accident, but the total plasma proteins were only 7.26 per cent. The blood sugar at this time was 176 mg. per hundred cubic centimeters. The fact that the value of the plasma proteins did not rise parallel to the degree of concentration of blood can be explained only on the basis of altered per-

meability of the capillary walls. The analysis showed that whole plasma was lost, rather than simply the water content.

The question may be raised whether the altered permeability involves all the capillaries of the body or only those employed in the process of forming the inflammatory exudate at the site of the burn. Hartman [16] has demonstrated that there is an increased amount of epinephrine in the circulation after extensive burns. It has been shown further by Petersen, Levinson and Hughes [17] that epinephrine increases endothelial permeability, and later lessens it. It would seem that concentration of the blood may be best explained on the basis of loss of plasma throughout the capillary bed of the body, as in traumatic shock, rather than on the basis of loss of fluid at the site of the lesion.

The changes just mentioned occurred primarily during the first twenty-four hours. The subsequent alterations noted may be best explained as phenomena of dilution.

CONCLUSIONS

1. In extensive burns there is first an increased permeability of the capillaries, which explains the characteristic concentration of blood in such cases.

2. There is no evidence to suggest that this alteration in the capillary walls persists during the period of toxemia. The late changes in the plasma proteins are probably phenomena of dilution.

3. The plasma fibrin and globulin show a characteristic rise, and the albumin shows a fall in concentration in burns.

REPORT OF CASES

CASE 1.—E. F., a white man, aged 18, was admitted to the hospital on March 23, 1926, for treatment of burns which resulted from an explosion of oxygen. There were second degree burns of both hands and forearms. The face, ears and neck were burned to the second degree.

CASE 2.—C. S., a white man, aged 33, was admitted to the hospital on May 15, 1926, for treatment of burns which resulted from an explosion of gasoline. There were second degree burns of the face, neck and hands.

CASE 3.—H. G., a white man, aged 39, was admitted to the hospital on May 18, 1926, for treatment of burns which resulted from an explosion of gas. There were second degree burns of the entire face, neck and both forearms.

CASE 4.—K. S., a white man, aged 30, was admitted to the hospital on June 5, 1926, for treatment of burns which resulted from an explosion of tar. There were second degree burns of the face, neck, left hand and forearm.

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16. Hartman, F. A.; Rose, W. J., and Smith, E. P.: Influence of Burns on Epinephrin Secretion, Am. J. Physiol. **78**:47, 1926.

17. Petersen, W. F.; Levinson, S. A., and Hughes, T. P.: Studies in Endothelial Permeability. I. The Effect of Epinephrin on Endothelial Permeability, J. Immunol. **8**:323, 1923.

CASE 5.—O. P., a white man, aged 22, was admitted to the hospital on Aug. 3, 1926, for treatment of a burn from gasoline. There were second and third degree burns on the left side of the chest and the left flank, extending from the seventh rib to the crest of the ilium. The burned area began at the midline posteriorly and crossed the midline anteriorly. This area was surrounded by a margin about 5 inches (12.7 cm.) wide which was burned to the first degree.

CASE 6.—W. R., a white man, was admitted to the hospital on Aug. 4, 1926, for treatment of burns which resulted from an explosion of gas. There were second degree burns of the left arm, hand and face.

CASE 7.—Z. T., a white man, aged 25, was admitted to the hospital on Sept. 3, 1926, for treatment of burns received when his clothes became ignited. There were third degree burns of the right side of the neck, and on the upper right forearm to the shoulder, involving the anterior, posterior and lateral surface. This extended into the axilla and then downward over the chest to the sixth rib. There were third degree burns of the left leg extending from the ankle to the knee. There was a similar third degree burn of the right leg. The remaining skin of both legs, from the toes up over the buttocks and involving the scrotum and perineum, was burned to the second degree. There were third degree burns over the sacral region and lower part of the back. There were also second degree burns of the left hand and arm. The cutaneous area involved was approximately 50 per cent of the total surface of the body.

CASE 8.—W. H., a white man, aged 22, was admitted to the hospital on July 15, 1926, for treatment of burns which resulted from an explosion of gasoline. There was a third degree burn involving the entire posterior aspect of the lower part of the left leg. The anterior and lateral aspects of the leg were burned to the second degree.

CASE 9.—W. C., a white man, aged 29, was admitted to the hospital on July 9, 1926, for treatment of burns which resulted from an explosion of gasoline. The entire right hand revealed a second degree burn. There was also a second degree burn of the lateral aspect of the right leg which involved about forty square inches.

CASE 10.—A. Y., a white man, aged 32, was admitted to the hospital on July 9, 1926, for treatment of burns which resulted from an explosion of gasoline. The entire left hand and the anterior aspect of the left leg were burned to the second degree.

THE CAROTID VENOUS PLEXUS AS THE PATH OF INFECTION IN THROMBOPHLEBITIS OF THE CAVERNOUS SINUS

ITS RELATION TO RETROPHARYNGEAL AND SPHENOIDAL SUPPURATION *

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SYNOPSIS

This communication is primarily to call attention to: (1) a type of cavernous sinus thrombophlebitis of otitic origin, which, although far from uncommon, has not received due consideration; how diagnosed, and what surgical steps may be taken for its relief, for successful surgery depends on early diagnosis; (2) the part (*a*) an associated sphenoidal sinus suppuration or (*b*) nasopharyngeal abscess plays in the production and diagnosis of a thrombophlebitis of the cavernous sinus of otitic or jugular vein origin.

Cavernous sinus thrombophlebitis of otitic origin is relatively not infrequent. Brunner [1] records that in twenty-two autopsies performed on patients who had died from generalized aural sepsis (in whom cavernous sinus disease was not suspected) twelve showed a septic clot in the cavernous sinus, the route of invasion of two of the twelve being by way of the carotid venous plexus (as demonstrated microscopically). I have personally treated thirty-two patients suffering from cavernous sinus infection (there were six recoveries, five of which were operative and one spontaneous). Fifteen complete autopsies were made, in six of which the infection had originated from aural suppuration; and an additional patient, although the infection was of pterygomaxillary fossa origin, had a suppurative otitis prior to death (case 2).

The schematic drawing (fig. 1) depicts the intracranial portion of the internal carotid artery (*I. C. A.*) and the plexus of veins surrounding it (the carotid venous plexus). The latter communicates with the jugular bulb posteriorly (*d*). Anteriorly the plexus communicates with the cavernous sinus, the ophthalmics (*Op. V.*) and the small veins which pass through the foramen lacerum and foramen ovale from the pterygomaxillary fossa (*a*). All unite to form a "venous lake" at the anterior extremity of the cavernous sinus. The chart also emphasizes

* Presented in part at the Fifty-Ninth Annual Meeting of the American Otological Society at Montreal, Canada, June 3, 1926.

1. Brunner, H.: Contribution to the Knowledge of Otogenic Cavernous Phlebitis, Monatschr. f. Ohrenh. **60**:2, 1926.

the communication of the carotid venous plexus with (*b*) the pharyngeal vault, and (*c*) the tympanic cavity, as well as the anterior venous communications of the cavernous sinus, and the ophthalmic with the veins of the pharynx; and their relationship to the retropharyngeal spaces, the pterygomaxillary fossa and the sphenoidal sinus mucous membrane.[2]

ROUTES OF INVASION OF CAVERNOUS SINUS FROM AURAL SUPPURATION

There are two routes by which cavernous sinus thrombophlebitis of otitic origin may arise.

(*A*) *Posterior (Petrosal) Involvement of the Cavernous Sinus.*—Middle ear infection usually extends from the jugular bulb through

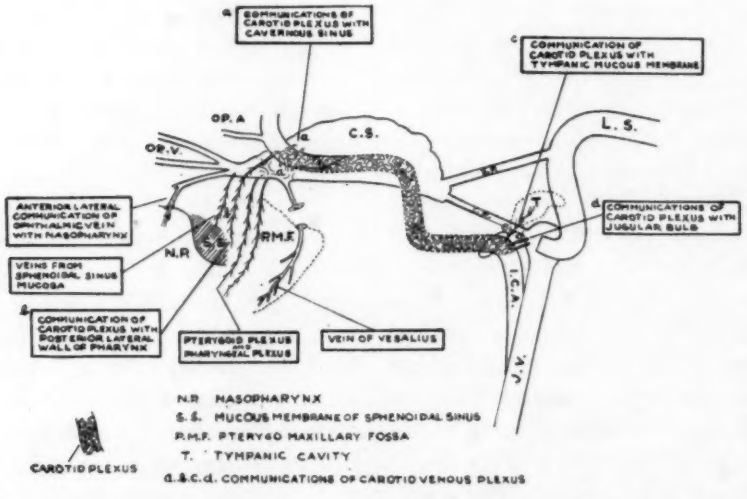

a COMMUNICATIONS OF CAROTID PLEXUS WITH CAVERNOUS SINUS
c COMMUNICATION OF CAROTID PLEXUS WITH TYMPANIC MUCOUS MEMBRANE
OP. A
OP. V.
C. S.
L. S.
ANTERIOR LATERAL COMMUNICATION OF OPHTHALMIC VEIN WITH NASOPHARYNX
d COMMUNICATIONS OF CAROTID PLEXUS WITH JUGULAR BULB
N.R
R.M.F.
VEINS FROM SPHENOIDAL SINUS MUCOSA
b COMMUNICATION OF CAROTID PLEXUS WITH POSTERIOR LATERAL WALL OF PHARYNX
PTERYGOID PLEXUS AND PHARYNGEAL PLEXUS
VEIN OF VESALIUS
I.C.A.
J.V.
N.R NASOPHARYNX
S.S. MUCOUS MEMBRANE OF SPHENOIDAL SINUS
R.M.F. PTERYGO MAXILLARY FOSSA
T. TYMPANIC CAVITY
a.b.c.d. COMMUNICATIONS OF CAROTID VENOUS PLEXUS
CAROTID PLEXUS

Fig. 1.—Schematic drawing of the communications of the carotid venous plexus.

the inferior petrosal to the cavernous sinus; much less frequently, from the knee of the sigmoid by the superior petrosal. This group of cases is fairly well recognized.

With a petrosal sinus origin, the clot forms in the posterior portion of the cavernous sinus, from which it extends forward. Thus time is given for a readjustment of the venous circulation from the orbit prior to the involvement of the anterior extremity of the cavernous sinus; consequently, exophthalmos or chemosis is not produced, at least, not until an ophthalmic vein becomes occluded—which is generally in the

2. Piebrantoni, L.: Vascular Relations Between Pharyngeal Vault and the Cranial Cavity, Arch. ital. d. otol. **36**:626, 1925.

terminal stage. It is the absence of early exophthalmos and chemosis which is responsible for the frequent failures to diagnose cavernous sinus suppuration in aural cases.

Clinical Diagnosis of Petrosal Cases: When the infection passes through a petrosal sinus, symptoms referable to one or the other of the branches of the fifth nerve—such as pain behind the eye or in the teeth—may be present. In many cases the patients' teeth have been removed for the relief of pain, when, in reality, edema of the nerve from infection of the adjacent sinus was the cause of this pain (case 2).

External rectus palsy may be an early symptom, the result of compression of the sixth nerve by the inflamed petrosal sinus as it passes under the sphenopetrous ligament. The third or fourth nerves or both may also be paralyzed by infiltration from the cavernous sinus during their course through it.

In chronic cases, exophthalmos, when it occurs at all, is likely to be a late manifestation due to the extension of the suppurative process into one of the ophthalmics, or the result of an orbital abscess. If, however, the phlebitis is fulminating, early chemosis and exophthalmos from venous occlusion may occur; but in the chronic pyemic cases, with great remissions of temperature and gradual clot formation, chemosis and exophthalmos do not occur.

(*B*) *Anterior* (*Carotid Plexus*) *Cases.*—The second route by which an otitic suppuration may reach the cavernous sinus is through the small venous plexus which surrounds the carotid artery. In this type, the infection ascends through the carotid canal, and, as the carotid plexus communicates with the anterior portion of the cavernous sinus, chemosis and exophthalmos are usually early, though transient, symptoms.

Clinical Types of Carotid Plexus Cases: Clinically, there are two types of carotid plexus invasion of the cavernous sinus of otitic origin: (1) cases associated with chronic aural suppuration, in which the thrombophlebitis of the plexus is secondary to caries of the bony covering of the carotid canal, situated in the anterior quadrant of the middle ear, which forms the posterior wall of the eustachian tube;[3] (2) cases which follow an acute aural suppuration, the phlebitis reaching the carotid plexus either by (*a*) a dehiscence in the floor of the tympanic cavity, (*b*) an osteophlebitis of a tympanic communication of the carotid plexus, or (*c*) secondary to a jugular bulb phlebitis.

Having once involved the small plexus surrounding the carotid artery, the phlebitic process is more likely to be transmitted to the cavernous sinus, because of the pulsating movement of the artery.

Another anatomic peculiarity is that infection by the carotid plexus attacks the cavernous sinus at its anterior extremity through the "venous

3. Alexander, G.: Otogenic Thrombophlebitis of the Cavernous Sinus, Monatschr. f. Ohrenh. **59**:971, 1925.

lake," which is made up of the ophthalmics, the veins from the nasopharynx and pterygomaxillary fossa, and the carotid plexus (*a* and *b* of scheme). Partial occlusion of the anterior end of the cavernous sinus, which causes a sudden associated disturbance of the return circulation, gives rise to slight exophthalmos and chemosis at an early stage in the disease. However, as the venous communications of the orbit are free, the proptosis and chemosis rapidly disappear with the reestablishment of the venous return. Later, there may be a recurrence of the exophthalmos and chemosis from a forward extension of the thrombophlebitis in the ophthalmics, with or without an orbital abscess.

Diagnosis of Carotid Plexus Cases: (*a*) A suppurating ear associated with signs of acute sepsis and slight chemosis and exophthalmos should suggest that the cavernous sinus has been infected by way of the carotid plexus.

(*b*) A chronic otitic sepsis (slight rise in temperature and sweating, or severe remissions) associated with the operative findings of caries and granulations in the anterior quadrant of the tympanic cavity during operation makes a thrombophlebitis of the carotid plexus a possible diagnosis.

Granulations on the carotid artery, disclosed during a radical operation performed for the cure of suppuration alone (unassociated with signs of sepsis), should not be curetted. The trauma of their removal was followed by a carotid plexus thromophlebitis in one of my early cases.

RELATION OF RETROPHARYNGEAL SUPPURATION TO DISEASE OF THE CAVERNOUS SINUS

The significance of a retropharyngeal abscess in the diagnosis of cavernous sinus thrombophlebitis in the presence of aural suppuration *after infancy* has not been sufficiently emphasized, although an examination of the literature disclosed a considerable number of cases. The postpharyngeal suppuration originates from retrograde thrombophlebitis of the veins communicating with the cavernous sinus and the pharynx, and is likely to pass unrecognized, because its location in the lateral wall of the superior portion of the nasopharynx—that is, above the palate—prevents the swelling from being seen during oral examination. Consequently, (*c*) a retropharyngeal or a pterygomaxillary fossa abscess in the course of an aural sepsis in a patient over 18 months of age should suggest that the cavernous sinus is involved.

CASE 1.—*Acute otitic pyemic sepsis with early involvement of the cavernous sinus by the carotid plexus associated with a secondary ptergomaxillary fossa abscess.*[4]

4. Detailed history, in Eagleton, W. P.: Cavernous Sinus Thrombophlebitis and Allied Septic and Traumatic Lesions of the Basal Venous Sinuses, The Macmillan Company, 1926; French translation, Masson et Cie.

The accompaning temperature chart resembles the excursions of lateral sinus thrombosis (fig. 2).

Hisiory.—A boy, aged 6, had had an earache, followed by delirium and a chill. A paracentesis was performed. From an early stage of the condition the pulse rate was disproportionately accelerated (showing profound intoxication). On the seventh day of the disease the mastoid was explored; little pathologic change was found. The day after the operation the child had slight exophthalmos with chemosis, which had disappeared on the following day. (Although their diagnostic significance was not appreciated, the exophthalmos and chemosis were indicative that the infection had passed through a dehiscence in the floor of the tympanum into the jugular bulb, and ascending through the carotid plexus had involved the cavernous sinus as in no other way could a proptosis be produced as early as the eighth day after the original earache.)

The temperature continued to be pyemic, but the patient did not have chills. Blood cultures were negative throughout. The lateral sinus was explored, but no clot or phlebitis was demonstrated.

On the sixteenth day the child had a *recurrence of the exophthalmos* with slight external ocular paralysis — ptosis and double vision. A diagnosis of "cavernous sinus thrombosis from jugular bulb phlebitis" was then made.

The carotid artery was ligated and the jugular bulb opened; the latter contained free pus. The *exophthalmos and chemosis immediately disappeared and did not recur,* although the child lived for several weeks.

A second chill occurred on the twentieth day of the disease. On the twenty-fifth day the patient developed *difficulty in swallowing, but did not complain of pain. Nothing could be seen in the throat, but a soft mass could be felt in the upper posterior lateral angle of the nasopharynx* (to which little attention was paid because of the absence of pain and the difficulty in breathing).

Death occurred from a terminal meningitis.

Autopsy (Dr. John V. Bissett).—The petrosal sinuses were normal; and the infection in the jugular bulb had been controlled by drainage. The upper end of the carotid canal and the cavernous sinus of the homolateral side were bathed in free pus. The infection also involved the opposite cavernous sinus. The superior portion of the nasopharynx contained an abscess beneath the mucous membrane which filled the pterygomaxillary fossa; the upper boundary of the abscess was formed by the base of the skull, with the foramen lacerum as its center. The abscess apparently had developed from a thrombophlebitis of the veins passing from the cavernous sinus into the nasopharynx through the lacerum. *The sphenoidal sinus contained free pus.*

INFLUENCE OF CHRONIC SPHENOIDAL SUPPURATION IN CAUSATION OF DISEASE OF THE CAVERNOUS SINUS

A *previous,* though quiescent, infection of the sphenoidal mucous membrane may be a causative factor in the production of septic phlebitis of the cavernous sinus when there is superadded another infection—acute otitis media, tonsillitis, or sinusitis—or severe trauma from operative procedure (such as enucleation of tonsils or sphenoidal sinus exenteration). This has not hitherto been emphasized, although O'Malley [5] observed that in all cases of death from disease of the

5. In discussion of paper by Davis, H. J.: Proc. Roy. Soc. Med., 1912.

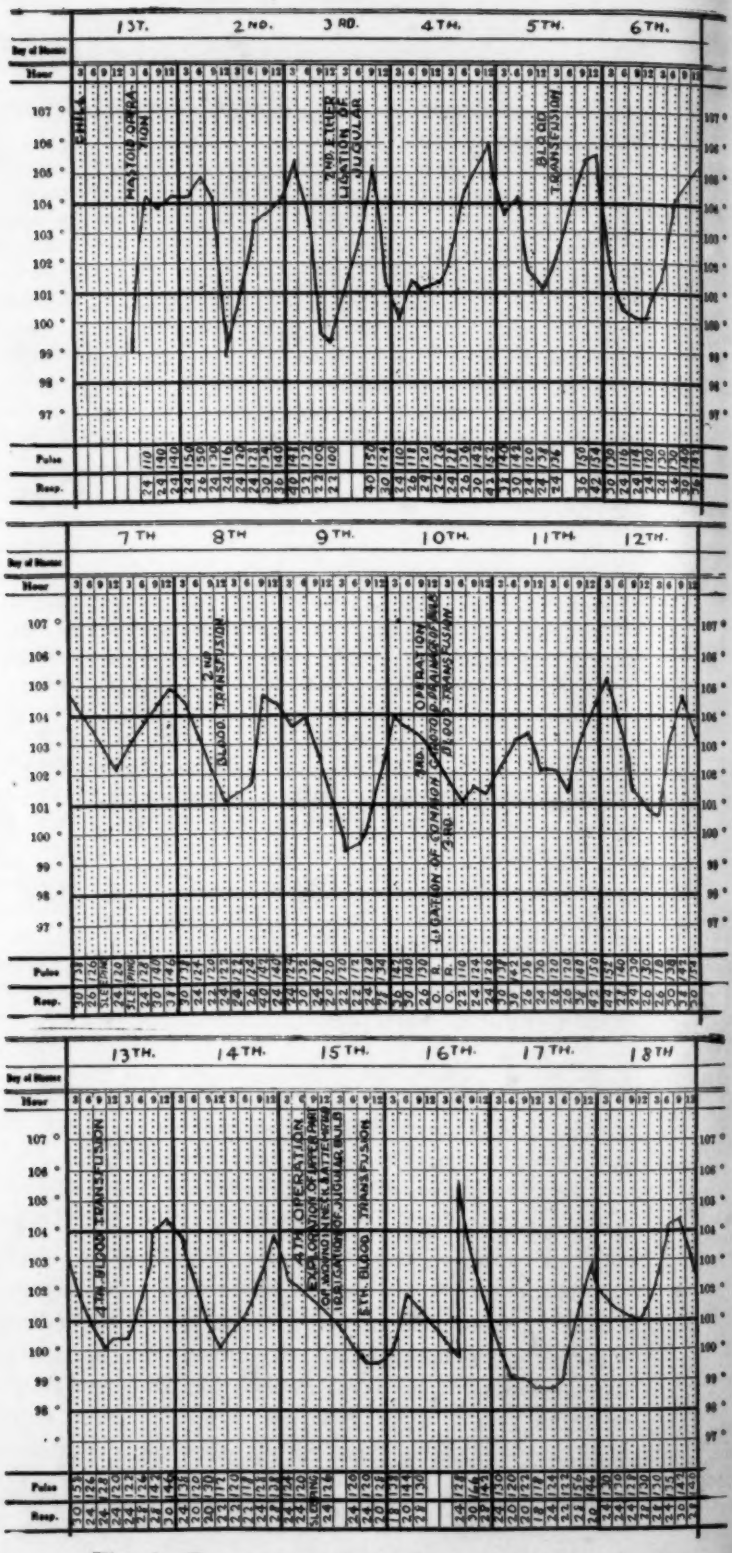
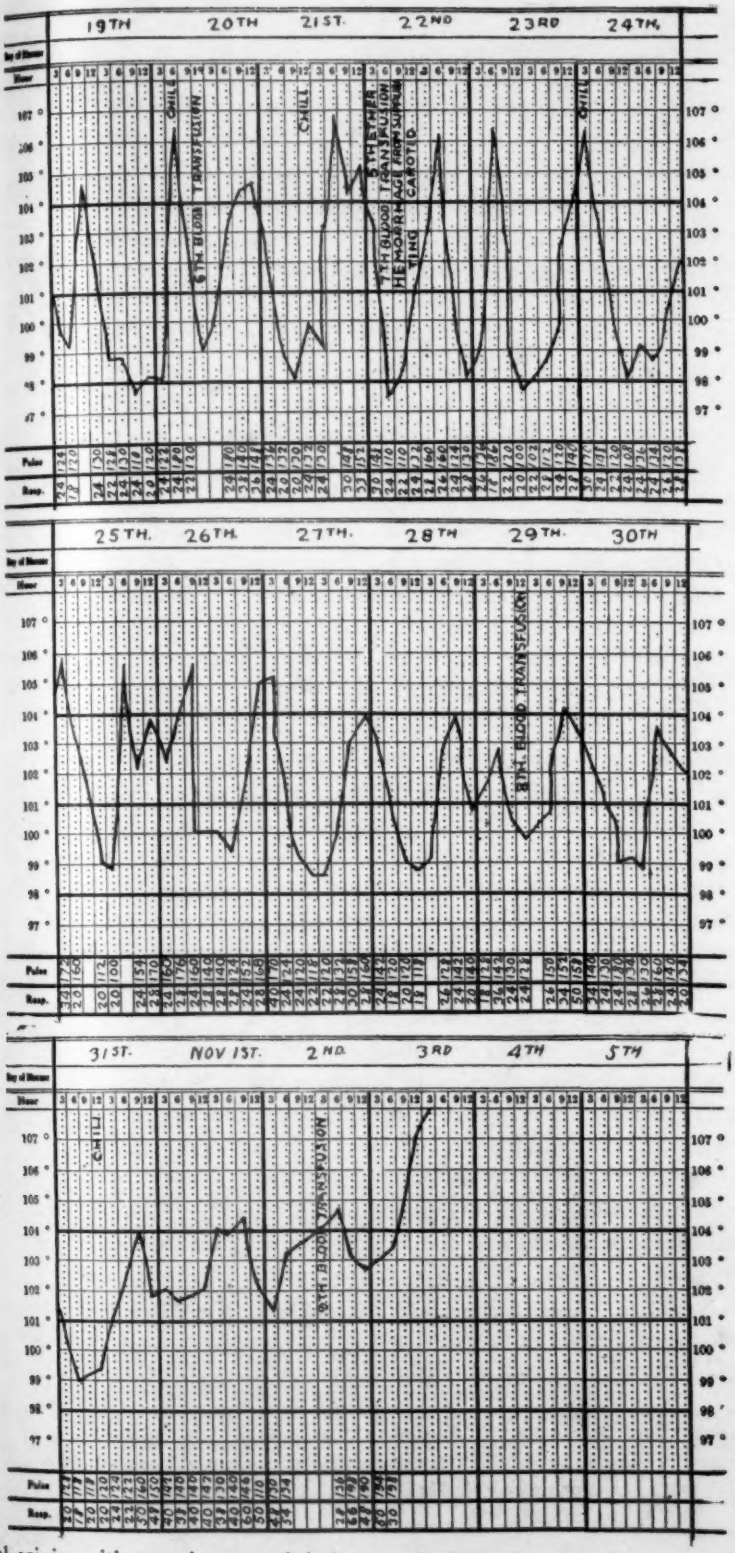


Fig. 2.—Temperature chart of case of abscess in cavernous



ural origin, with transient exophthalmos and chemosis (case 1).

cavernous sinus following a tonsillectomy the sphenoid had been involved.

On the other hand, Turner and Reynolds [6] have demonstrated microscopically that the sphenoidal suppuration which accompanies cavernous sinus disease (as in the case herein reported) is secondary to the infection of the cavernous sinus, the sphenoidal suppuration being due to nutritional death of (submucous, and osseous) tissue the result of a thrombophlebitis which descends from the cavernous sinus into the sphenoid.

An existing but quiescent inflammation of the sphenoid may be a causative factor in the production of a cavernous sinus infection because: (1) The sphenoid is the only accessory sinus in anatomic proximity to a large venous radicle in which the circulation is free, but slow, with many eddies; this is favorable to the production of a septic thrombophlebitis. (2) In suppuration of the sphenoidal mucosa, the infection is separated from the cavernous sinus by a thin layer of compact bone especially liable to nutritional death from circulatory disturbance. (I have found "discoloration and roughness of the intracranial surface of the sphenoid" to be a usual postmortem condition in cavernous sinus infection). (3) The sphenoid is lined with a delicate mucous membrane (normally easily detached from the bone). (4) Finally, both the mucous membrane and bone are perforated by veins (the sphenoidal plexus) which empty into the cavernous sinus. Consequently, when a sphenoidal sinus is the seat of a chronic purulent inflammation, accompanied as it must be by thrombosis of many of its small veins, and later is attacked by an acute intercurrent infection, its veins are especially likely to become the avenue of an infection to the cavernous sinus, since the superimposed acute phlebitic process passes through only a few millimeters of thrombosed tissue before it enters directly into the large venous radicle.

The cavernous sinus, because of its relationship to the veins of the sphenoid sinus, lacks the protection afforded to infection from the pterygoid and pharyngeal plexus—a long course through dense mucous and submucous tissue muscle, fascia and bone of the pharyngeal vault and base of the skull. The anterior extremity of the cavernous sinus thus possesses anatomic and physiologic factors favorable to the intracranial extension of venous infection which are not found in any other region of the head.

Influence of Trauma.—If such be the fact, it is evident that (1) all unnecessary operative trauma should be avoided (at least for a considerable period) whenever the history warrants the suspicion that a previous

6. Turner, A, Logan, and Reynolds, F. Esmond. Furuncle of the Right Nasal Vestibular; Septic Thrombosis of the Cavernous Blood Sinuses; Leptomeningitis. Death; Autopsy, J. Laryng. & Otol. **41**:73 (Feb.) 1926.

sphenoidal sinus infection was associated with an osteophlebitis of the sphenoidal vein—pain on top of the head or behind the eyes, and blurred vision accompanied by a persistent nasopharyngeal discharge.

(2) An associated suppurative process—acute otitis media, tonsillitis or sinusitis—should be regarded with apprehension if accompanied by the slightest symptoms of cavernous sinus disease.

CASE 2.—*Chronic infection of the pterygomaxillary fossa (of dental origin) ascended as a thrombophlebitis and entered the cranium by the foramen ovale, giving rise to (1) pus in both cavernous sinuses, (2) extradural abscess, (3) localized meningitis of the floor of the middle fossa and (4) a brain abscess in the anterior portion of the temporosphenoidal lobe. Later, the phlebitic process retrograded through the carotid plexus and occasioned (5) a jugular bulb and sigmoid sinus suppuration and finally (6) a purulent otitis and mastoiditis, the latter probably occurring from an osteophlebitis of the veins of the floor of the middle ear and mastoid.*

Previous History.—About nine months before he was admitted to the hospital, the patient, a man, noticed that the skin on his face and body was becoming yellow. This condition was accompanied by a feeling of general weakness. One week later, he suffered from pain in his teeth and a slight swelling of the right side of his face. Two upper back teeth were removed. Following the extraction he could not open his jaw. He remained in bed for two weeks. Later, the jaw was curetted. In spite of continued dental treatment, he gradually grew weaker. The swelling of the face subsided somewhat, but he began to have pain in his lower teeth; this was relieved by an extraction. During the next month, although able to work, he consulted several physicians because of the swelling and pain over the right side of the upper jaw. One physician diagnosed the condition as "gumma of the jaw." In February, seven months after the beginning of his illness and two months before admission to the hospital, the patient was hit on the swollen part of the jaw. From this time, the pain, edema and illness increased.

On April 20, he was admitted to the Newark City Hospital. He was conscious but "dopy," complained of pain in the jaw and dizziness; and had the low, irregular temperature of chronic sepsis.

Examination.—The right side of the face and eye were swollen. The swelling resembled that of an abscess under the temporal muscle; however, it had a sharp line of demarcation as if the pressure was under the parotid gland rather than under the temporal muscle. It did not extend downward into the neck or involve the lower jaw. The edema of the lids of the right eye was so great that the eye could not be seen, but forcible retraction showed that there was no proptosis. The mouth could not be opened.

The blood count showed red cells, 4,800,000; leukocytes, 13,000; polymorphonuclears, 75 per cent; lymphocytes, 25 per cent. Repeated blood cultures were negative.

Diagnosis.—The condition was diagnosed as "pterygomaxillary-fossa infection subsequent to infiltration of the dental canal from the extraction of teeth, with acute exacerbation developing seven months later from trauma, followed by an osteomyelitis and a protective meningitis of the floor of the middle fossa from an ascending thrombophlebitis." (The diagnosis was based on (1) the history, (2) the limitation of the swelling, (3) the inability to open the jaw, (4) the presence of chronic sepsis and (5) the mental state of the patient.)

Operation was advised. This was to consist of a "radical exploration of the pterygomaxillary fossa and base of skull; to be preceded by ligation of the carotid plexus and possibly associated with injury to the facial nerve, if the infective process in the bone could not otherwise be fully eliminated." (This major procedure was suggested because I had seen a patient with a similar chronic pterygomaxillary-fossa infection which seventeen skin incisions for the evacuation of pus had failed to control.)

Subsequent History.—The temperature continued to be irregular and slightly elevated. The patient complained of a constant pain in the face and the side of the head; but he became rational and alert. The swelling of the side of the head and face having extended upward into the temporal region, on May 6, a skin incision was made which evacuated a quantity of pus from under the temporal muscle. An incision below the angle of the jaw was performed on May 12, and another one was made on June 3, each time liberating a considerable amount of pus and followed by a marked subsidence of the swelling and relief from pain. The chronic septic condition continued, however, associated with a loss of flesh.

On June 10, he was unable to move his eye outward because of right external rectus paralysis (which may have been present for some time as previously the swelling of the lids prevented an inspection of the ocular movements).

The patient had complained of slight pain in the right ear; on June 13, there was profuse discharge with a perforated drum membrane.

On the morning of June 21, the patient was slightly dazed, but was able to answer all questions intelligently and promptly. Suddenly he had repeated convulsions, characterized by "nystagmus to the left, a spasticity of the left arm and leg, with complete flaccidity of the right arm and leg." A lumbar puncture was made; the fluid was clear and under moderate pressure; the number of cells had not increased and no organisms were found in the smear or on the culture.

(Following the convulsions he again became rational and alert.)

On June 25, it was decided that if the patient could be put into condition for an operation, the pterygomaxillary fossa should be entered (going across the base of the skull) and an effort made to combat what undoubtedly seemed to be a pterygomaxillary abscess, with secondary thrombophlebitis of the veins passing either through the foramen lacerum or ovale. The thrombophlebitis had occasioned an osteomyelitis of the base of the skull, with an extradural abscess, a localized meningitis or a brain abscess.

Death followed a blood transfusion prior to the operation.

Autopsy (Dr. Harrison S. Martland).—Soft Parts: The reflection of the scalp revealed extensive edema of the right temporal muscle with pus, which came from below, between the muscle and bone.

Nasopharynx: The inflammatory infiltration extended across the base of the skull into the *pterygomaxillary fossa, internal to which on the posterior pharyngeal wall* of the right side (between it and the anterior body of the vertebrae) there was *a large amount of pus.*

This condition existed, although the only difficulty in swallowing that the patient had was attributable to the injury and external swelling. The only complaint of dysphagia was recorded on June 6 in the nurses' record: *"fluids taken fairly well but difficulty in swallowing."*

Bones of Base of Skull: The base of the skull did not contain microscopic evidence of caries or necrosis. There was a purulent extradural exudate on

the inner half of the floor of the right middle fossa, which contained considerable pus; the center of the purulent area was over the fossa ovalis.

Basal Sinuses: The right and left cavernous sinuses contained free pus. There was a thin, purulent exudate on the subdural surface of the left middle fossa in the region of the cavernous sinus. There was purulent thrombophlebitis of the right jugular bulb, while the descending portion of the right sigmoid contained a clot. (The infection apparently entered by the veins of the ovale, involved the right cavernous sinus, and descended, by way of the carotid plexus, to the jugular bulb, as the petrosal sinuses were not infected.)

Meninges and Brain: The removal of the brain revealed numerous adhesions of the tip of the right temporal sphenoidal lobe. The pia-arachnoid of this area was slightly brownish. The breaking of the adhesions revealed an abscess in the brain substance, the adhesions to the floor of the right middle fossa forming its stalk and walling it off from the rest of the meninges. The abscess cavity was from about ¾ to 1 inch (1.8 cm. to 2.5 cm.) in size and filled with greenish pus. It had a capsule, made up of a granulating membrane from 2 to 3 mm. thick and slightly red. The cavity of the abscess was situated below the ventricle and the caudate nucleus, and extended to within a few millimeters of the descending horn of the right lateral ventricle. The brain tissue surrounding the upper part of the abscess was slightly yellow and edematous; the edema encroached on the corpora striata. The right lateral ventricle was somewhat compressed in the middle. In the overlying ependyma there were small punctate hemorrhages.

Evidence of general meningitis was not found.

The right mastoid contained some pus (a recent process, as the cells were not broken down).

The sphenoidal cells were free.

Comment.—From the history of the patient and the postmortem observations, it is evident that (1) the infection followed a therapeutic infiltration of a dental canal. After a prolonged period of activity it became semiquiescent but (2) underwent an acute exacerbation as the result of the trauma from a blow. From this time the patient suffered from (3) an abscess in the pterygomaxillary fossa associated with (4) thrombophlebitis.

The abscess (encased externally by the parotid gland and the fascia) dissected upward under the temporal muscle, downward into the neck, and forward into the cheek and was evacuated in all these regions; while the deeper pterygomaxillary collection (although causing trismus from infiltration of the pterygoid muscles) did not occasion difficulty in swallowing or interfere with respiration because of its high position.

The infective process was not controlled by the peripheral incisions because from the beginning it involved the venous radicles of the pterygomaxillary fossa; by them it entered the skull through the foramen ovale and invaded the right cavernous sinus; from the right it extended to the opposite cavernous sinus. The chief area of intravenous purulent accumulation was the lake at the anterior end of the right cavernous sinus; from it the thrombophlebitis traversed the carotid plexus to the jugular bulb, from which it retrograded into the sigmoid sinus.

A thrombophlebitis of one of the smaller radicles caused (1) the protective meningitis—as evinced by adhesions of the brain to the dura—and (2) the brain abscess with a capsule and stalk.

A few days before the death of the patient the phlebitic process manifested itself by a suppurative otitis, probably from a thrombophlebitis of the veins entering from the jugular bulb or carotid plexus, as necrosis of the bone was not found at autopsy. (This view is strengthened by the fact that a retropharyngeal abscess associated with mastoid involvement from direct extension through the base of the previous pyramid, although a not uncommon complication in infants by way of a dehiscence, is unknown in adults without microscopic evidence of necrosis or caries.)

PRINCIPLES OF SURGICAL TREATMENT OF CAVERNOUS SINUS INFECTION

(*a*) When there is a reasonable suspicion of cavernous sinus infection, the inflamed sinus should be placed at rest by fractional [7] or complete ligation of the internal carotid.

(*b*) Following this, the large venous radicle into which the smaller vein empties, or through which the primary or secondary area of infection has been transmitted, must be surgically opened.

(*c*) Nature's reparative processes must be aided—by repeated transfusions, preferably from an immunized donor.

Treatment.—Of chief importance is the early diagnosis that the infection *has probably reached the cavernous sinus;* but whether it ascended (*a*) by way of the petrosal sinuses (coming from behind; with symptoms of pain from involvement of the fifth nerve with ocular paralysis from the third and fourth nerve), or (*b*) by the carotid plexus (attacking the sinus from in front, thus causing transient exophthalmos) is of secondary consideration.

(1) Aid to Spontaneous Recovery: Although it is recognized that rest is necessary in nonpurulent phlebitic processes, the application of the principle in the treatment of septic phlebitis has not been sufficiently emphasized.

My experience would suggest that if, during an early stage of cavernous sinus infection—before the clot has broken down—the inflamed parts are placed at rest, and the primary focus radically removed, spontaneous recovery will result in a considerable proportion of cases. Rest of an inflamed cavernous sinus can be obtained only by stopping the pulsations of the intracranial portion of the internal carotid, as it is contained within the cavernous sinus. For I am convinced that any trauma of a vein which is the seat of a septic process—

7. Kerr, H. H.: Fractional Ligation of Common Carotid Artery in Treatment of Pulsating Exophthalmos, Surg. Gynec. Obst. **41**:565 (Nov.) 1925.

whether caused by injury during operation, forcible palpation during examination, movement from the contractions of adjacent muscles or the pulsations of an accompanying artery—not only tends to prevent Nature's control of the infection by clot formation, obliteration and canalization, but in the vast majority of cases is the deciding factor in the propagation and extension of the septic process itself.

(2) Indications for Opening the Cavernous Sinus: The foregoing cases and autopsies demonstrate that pus and broken-down clot usually are located only in the large venous sinuses, where the circulation is free but slow. The smaller venous tracts, such as the petrosals, the veins of Vesalius, or the carotid plexus (while frequenting the route of a transmitting retrograde thrombophlebitis), are anatomically adapted to a spontaneous cure by obliteration. Consequently, when there is clinical evidence that the cavernous sinus is the seat of a purulent collection—such as a continuation of pyemic sepsis after the jugular bulb has been opened, or when the infection has resulted from a retrograde thrombophlebitis from the nasopharynx (case 2)—the large venous lake itself should be opened.

(3) Surgical Approach to Sinus: The operative attack should follow the path of the infection as far as possible. However, if there is extreme exophthalmos—and a blind eye—the sinus can be adequately drained from in front by Mosher's [8] operation, after the common carotid has been ligated. The latter not only diminishes hemorrhage which is otherwise uncontrolled, but also puts the parts at rest.

If the infection has passed a petrosal sinus, the cavernous sinus may be opened along the floor of the middle fossa. All efforts to drain through the petrosals themselves (as advocated by Ballance [9]) have been unsuccessful. This is to be expected on anatomic and pathologic grounds, except possibly in the cases of a very large petrosal with free pus throughout its entire extent—a very rare condition.

If the infection has ascended by the carotid plexus, early carotid ligation with an exploration of the jugular bulb should be followed by an incision of the cavernous sinus through the middle fossa.

An associated *retropharyngeal* abscess may be drained through the mouth, but a *pharyngomaxillary* abscess must be evacuated externally.

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8. Mosher, Harris Peyton: The Orbital Approach to the Cavernous Sinus, Laryngoscope **24**:709-717, 1914.

9. Ballance, C. A., and Hobhouse, Neil: A Case of Septic Thrombosis of the Left Sigmoid, Cavernous and Inferior Petrosal Sinuses, a Suggestion for Treatment in Future Cases, Tr. Ninth Internat. Otol. Congress, Boston, 1912.

THE STATUS OF ENTEROSTOMY IN THE TREATMENT OF ACUTE ILEUS

A STATISTICAL INQUIRY *

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Any one who is interested in the treatment of patients with acute ileus must have been encouraged by the enthusiastic recommendation that has been given to enterostomy as a life-saving procedure. Within the past fifteen years, Elsberg,[1] Woolsey,[2] Bonney,[3] McGlannan,[4] McKenna,[5] McKinnon,[6] Richardson,[7] Summers,[8] DeLore and Conrozier,[9] Wortman,[10] Vollhardt,[11] Walker,[12] C. H. Mayo,[13] Barber,[14] Boit,[15] W. J. Mayo,[16] Taylor [17] and many others have recommended the use of enterostomy, particularly of a high jejunostomy. Some of these observers have reported cases to support their recommendations; a few have reported series of cases, while others have merely enthusiastically recommended jejunostomy. Since some of these recommendations were authoritative, we were inclined to accept them, but certain facts have led us to question the procedure. For example, we found that there was a high mortality rate in the enterostomy group of a series of cases of

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* Read before the Section on Surgery at the Annual Meeting of the Medical Society of the State of New York, May 13, 1925.

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10. Wortman, W.: Med. Klin. **17**:932 (July 31) 1921.
11. Vollhardt, W.: Deutsche. Ztchr. f. Chir. **164**:352 (July) 1921.
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14. Barber, W. H.: Enteroparesis, J. A. M. A. **79**:1824 (Nov. 25) 1922.
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acute ileus at the Presbyterian Hospital, from 1914 to 1923, inclusive. Moreover, the mortality rate in a similar group at the Roosevelt Hospital, from 1910 to 1919, inclusive, agreed closely with the results at the Presbyterian Hospital. We examined the recent literature on acute ileus and compiled as many statistics of case series as we could find, up to 1924. However, the actual statistics do not seem altogether to support the claims of those who have recommended enterostomy. We have not been able to discover definite, incontrovertible proof that enterostomy has lowered the mortality rate in cases of acute ileus

The evidence furnished by any small group of patients on whom enterostomy had been performed is not of any definite value unless it concerns all of the patients in that particular series of cases of acute

TABLE 1.—*Groups of Enterostomy Cases**

Date Published or Years Covered	Name of Author or Hospital Reporting	Number of Cases	Number of Deaths	Mortality Percentage
Published 1913... (1893-1912, inc.)	A. McGlannan ..... Johns Hopkins Hospital St. Agnes Hospital	81	40	49.4
Published 1920...	E. A. Codman.....	12	7	58.3
Published 1920... (1910-1919, inc.)	E. P. Richardson..... Massachusetts General Hospital	36	18	50.0
Unpublished..... (1910-1919, inc.)	The Roosevelt Hospital, New York City..	47	37	78.7
Published 1921... (1911-1920, inc.)	J. M. T. Finney..... Johns Hopkins Hospital Union Memorial Hospital	48	29	60.4
Published 1921...	W. Wortman	35	12	34.3
Unpublished..... (1914-1923, inc.)	The Presbyterian Hospital, New York City	90	69	76.6
Total		349	212	
Average mortality of collected cases.....				60.7

* These groups are isolated from the series collected in table 2.

ileus who received similar treatment. Even then, the evidence is inconclusive because of the great variations that occur in the mortality rate of small series. At the Presbyterian Hospital we found one group of eleven consecutive patients on who enterostomy was performed in which the mortality rate was only 10 per cent, and another similar group with a mortality rate of 90 per cent. Nevertheless, we have included in our enterostomy table one small group because it appeared to be complete and was published as a part of a series by an observer of recognized conservatism. A comparison of tables 1 and 2 show that the average mortality rate for patients wtih acute ileus on whom enterostomy is performed is 19 per cent higher than that for the entire series in the present compilation. Further comparison shows that in every series quoted, the mortality rate of the enterostomy group is higher than that of the series. A comparison of the mortality rate of the enterostomy group with that of the nonenterostomy group is even more striking and less favorable to enterostomy (table 3).

There must be some explanation for this disagreement between what appear to be the facts and what are believed to be the facts by a considerable number of competent observers. The basis for the belief that enterostomy is effective in the treatment of acute ileus is subjective, and therefore to be mistrusted. One fact is not compared with another fact, but rather a fact is compared with a supposition. The patient who is expected to die recovers after an enterostomy has been performed, and the enterostomy is believed to have been responsible for the recovery.

TABLE 2.—*Series of Cases of Acute Ileus**

Date Published or Years Covered	Name of Author or Hospital Reporting	Number of Cases	Number of Deaths	Mortality Percentage
Published 1913... (1893-1912, inc.)	A. McGlannan ..... Johns Hopkins Hospital St. Agnes Hospital	181	73	40.3
Published 1920...	E. A. Codman.....	41	14	34.1
Published 1920... (1908-1917, inc.)	E. P. Richardson..... Massachusetts General Hospital	118	49	41.5
Unpublished..... (1910-1919, inc.)	The Roosevelt Hospital, New York City	186	88	47.3
Published 1921... (1911-1920, inc.)	J. M. T. Finney..... Johns Hopkins Hospital Union Memorial Hospital	217	76	35.0
Published 1921...	W. Wortman	172	55	31.9
Unpublished..... (1914-1923, inc.)	The Presbyterian Hospital, New York City	174	101	58.0
Total		1,089	456	
Average mortality of collected cases.....				41.8

* The enterostomy groups collected in table 1 are included in these series.

TABLE 3.—*Comparison of Mortality in Acute Ileus. Series Compiled in 1900 and in 1925**

1,000 cases compiled by C. D. Gibson, published in 1900
1,089 cases compiled by the present writers in 1925

Cases Referred to 1900 and 1925 Collections	Number of Cases		Mortality Percentage	
	1900 Series	1925 Series	1900 Series	1925 Series
Entire series.....	1,000	1,089	43.2	41.8
Nonenterostomy group.....	810	740	38.2	33.0
Enterostomy group.....	190	349	64.2	60.7

* The reduction in mortality of nonenterostomy group is slightly greater than in the enterostomy group.

The fact is that the patient lived, and the supposition is that he would have died. Perhaps we are prejudiced in favor of enterostomy, knowing that in controlled animal experiment, evacuation and irrigation of obstructed loops is of proved efficacy. On the other hand, certain cases of acute ileus in which the patient was expected to die and in which recovery was made without enterostomy, are likely to be forgotten. In bedside experiments there is no control except the painstaking honest comparison of carefully checked statistics. We believe that a certain amount of the enthusiasm for enterostomy in the treatment of acute ileus is a result of a natural optimism unchecked by careful reasoning,

and of a failure to test theory against experience, but we cannot believe that this is the complete explanation.

If there is anything in our compilation of statistics to support the present enthusiasm for enterostomy, we have not discovered it. We had hoped to be able to show that enterostomy is an effective treatment; we are still inclined to believe that it is, but we find ourselves trying to explain why statistics do not show it.

At first one may be shocked to find the mortality rate of the enterostomy group of patients so much higher than that of the nonenterostomy group in the same series. In the series that Gibson collected, it was 26 per cent higher and in this series almost 28 per cent higher (table 3). In considering these statistics, enterostomy appears to be a dangerous

TABLE 4.—*Rise in Mortality Rate Due to Late Operation*

Taken from the Roosevelt Hospital Statistics, 1910-1919, inclusive.
Time between onset and operation was recorded in only 164 cases.

	Hours Between Onset and Operation				
Mortality Percentage	12	24	48	72	72+
90	..	..	..	..	..
80	..	..	..	..	.. 59
70	..	..	..	..	
60	..	..	..	.. 20	.
50	..	..	32		
40	..	..			
30	..	.. 25			
20	..				
10	.. 28				

Average mortality of all cases was 49.7 per cent. Numbers represent the number of cases in which operation was performed in the 12 or 24 hour period.

procedure. It must be kept in mind, however, that enterostomy has not been performed as a routine procedure. It was performed only in 19 per cent of Gibson's compiled series and in 32 per cent of the present compiled series. This operation has been reserved generally for those cases in which the patient is sickest or the disease is most advanced, or both (table 5, star footnote).

It is fallacious to compare the mortality rate of the entire enterostomy group with that of the entire nonenterostomy group in any series until it is determined whether each comprises the same percentage of late cases as does the other. It is recognized generally that the mortality rate of patients with acute ileus depends primarily on the time at which operation is performed rather than on the type of operation (tables 4 and 9). The entire enterostomy group includes about the same percentage of late cases as the entire nonenterostomy group of our series at the Presbyterian Hospital (table 5, dagger footnote), and, therefore (if other factors were equal), its mortality rate should be about the same. We have made a comparison of this enterostomy group with the nonenterostomy

group in table 6, and find that the mortality rate of the entire enterostomy group is considerably higher than that of the entire nonenterostomy group. The same fact has been noted in table 3 in which we compared the mortality of enterostomy and nonenterostomy groups having an unknown percentage of late cases. These observations tend to confirm each other. In table 7 we have compared the mortality of the late (72+ hour) groups of cases—enterostomy against nonesterostomy.

TABLE 5.—*Percentage of Enterostomy Cases in Each Time Group; From Presbyterian Hospital Statistics, 1916-1923, Inclusive*

Time Group, Hours	Total Cases in Time Group	Nonenterostomies†	Enterostomies	Percentage of Enterostomies in Each Group
12	2	1	1	50
12 to 24	16	7	9	56
24 to 48	20	14	6	30
48 to 72	23	12	11	48
72+	79	48	31	40
Totals	140	82	58*	

* Out of fifty-eight enterostomies only sixteen were performed within forty-eight hours after onset and thirty-one (more than half) were done on late cases.

† The nonenterostomy group has 73 per cent of cases after forty-eight hours. The enterostomy group has 72.4 per cent of cases after forty-eight hours.

TABLE 6.—*Comparison Between Mortality of Entire Enterostomy Group and Entire Nonenterostomy Group; Taken from the Presbyterian Hospital Statistics*

Of the series of one hundred and forty acute ileus cases (1916-1923, inc.) fifty-eight patients had enterostomies and eighty-two did not.

	Total Cases	Recovered	Died	Mortality
Entire enterostomy group	58	12	46	79.2%
Entire nonenterostomy group	82	44	38	46.3%

TABLE 7.—*Comparative Mortality of Enterostomy Late Group and Nonenterostomy Late Group; Taken from the Presbyterian Hospital Statistics*

The time group operated on more than forty-eight hours after onset comprised fifty-three cases (in the years 1920-1923, inc.). Of these, thirty patients had entefostomies and twenty-three did not.

	Total Cases	Recovered	Died	Mortality
Enterostomy group	30	12	18	60%
Nonenterostomy group	23	12	11	47.8%

Again, we find the former decidedly higher. This tends to confirm the previous observations. If entertostomy is as valuable as it is said to be, one would expect to find the mortality rate of the group so treated (other factors being equal) lower than that of the nonenterostomy group. However, the other factors have probably not been equal, and enterostomy has generally been performed with or in sequence to an exploratory or relief operation as well as on the sickest patients or on those with acute ileus in an advanced stage. The poor choice of cases and the additional shock of the added procedure may account largely for the higher

mortality rate. Certainly enterostomy has not infrequently been performed as a last resort on patients who were in a critical condition following a relief operation, and who would probably have died anyway. The mortality rate could have been credited to the nonenterostomy group had not the enterostomy been performed in the unjustified hope of saving the patient. Enterostomy has been similarly performed on almost moribund patients who were thought not be able to tolerate further severe surgical procedures. Since enterostomy has frequently been performed without proper appreciation of its limitations, an undeserved appearance of danger has been given to the procedure.

Enterostomy is not a "sure cure," nor is there any magic about it. Like other surgical procedures, it must be performed in the right way and at the right time to be effective. In cases in which the patient has acute ileus in an advanced stage, the condition is primarily chemical, and purely mechanical treatment cannot be effective. The ileus poison has presumably been absorbed into the blood and tissues. It would be as unreasonable to expect the condition of the patient to improve after enterostomy had been performed in these cases as to expect a person to recover who had already absorbed a lethal dose of any other poison. It is also unreasonable to add surgical shock to fatal chemical poisoning. The primary object of enterostomy in the treatment of acute ileus is to secure drainage. Even if enterostomy is performed in time but a drainage is not obtained, the procedure is without value. If drainage is not secured at the end of twenty-four hours, the enterostomy should be carefully investigated and if necessary revised; however, this is rarely done, and in cases in which the condition is far advanced there is often little time for it. We feel that the considerations cited tend to explain why the statistical evidence we have collected does not serve to support the general enthusiasm for enterostomy. It is interesting to find this discrepancy between the statistical evidence and the general belief in the efficacy of enterostomy; it is of some importance to record it, but of far greater importance to call attention to the fact that statistics such as those we have collected can never be used to demonstrate the efficacy of enterostomy (if it is actually efficacious) in lowering the mortality rate in acute ileus. The reason for this is that the time factor, which is of primary importance in the mortality rate of patients with acute ileus, has not been recorded, and cannot be taken into consideration.

GROUP COMPARISON

Improvement can be shown only by fair comparison of one series or group with another. A fair comparison of the results to determine the influence of one procedure or another on them cannot be made without taking the time factor into consideration, and the time factor cannot be

taken into consideration unless the statistics are so analyzed as to make this possible. For example, in comparing two series, the following points should be noted: (*a*) the number of cases in the enterostomy group and in the nonenterostomy group of each series, that is, each series should be divided into an enterostomy and a nonenterostomy group; (b) the percentage of early cases (operated on within forty-eight hours after onset) in each entire series; (*c*) the percentage of early cases in each enterostomy group; (*d*) the percentage of early cases in each nonenterostomy group; (*e*) the mortality percentage of each entire series; (*f*) the mortality percentage of each enterostomy group; (*g*) the mortality percentage of each nonenterostomy group. These determinations are required to check the time factor (early operation) and the technic factor (enterostomy or nonenterostomy) as they affect the results of the operation expressed in terms of the mortality rate. Only after having secured them is one in a position to make a fair comparison of the technical results or to estimate whether any degree of improvement may be due to enterostomy. A lower mortality rate in one series cannot be said to indicate better technical results unless it is shown that this series has the same percentage (approximately) of late cases as that with which it is compared. Neither can it be said that lower mortality rate in one series is due to the effects of enterostomy unless it is shown that the improvement was not due to the time factor nor to better technic in the nonenterostomy group. It cannot be asserted that lower mortality rates in one enterostomy group is due to better technic until the time factor has been discounted by a percentage comparison of the early cases in the two groups being compared.

On the other hand, if one can show that two series each comprised the same percentage of late cases, it is fair to say that the lower mortality rate of one is probably due to better technic. Again, if one can show that the time factor was the same in two series, as well as in their respective enterostomy and nonenterostomy groups, and if one can show also that there was no material improvement in the nonenterostomy group, the lower mortality of one series can properly be attributed to the beneficial effect of enterostomy.

To summarize: Each series of cases of acute ileus is divided into two groups—an enterostomy and a nonenterostomy group. If the mortality rate of the entire series is lowered (and that of the enterostomy group as well) the mortality rate of the nonenterostomy group must remain relatively unchanged (or be higher than before), else the improvement in the rate cannot be ascribed definitely to the enterostomy group. The two enterostomy groups should be analyzed (being compared) as to whether the patient has ileus in an early or advanced stage. If the group with the lower mortality rate contains a larger proportion of early cases

than does the other, the improvement must be ascribed rather to earlier treatment than to improved technic. Granted that early treatment brings better results, in comparing the general mortality of any two entire series, they must be analyzed for early and late cases. If the one with lower mortality rate comprises a greater proportion of early cases than the other, the improvement is ascribed again to early diagnosis rather than to any special procedure such as enterostomy. Therefore, in order to prove the efficacy of enterostomy in lowering the mortality rate in cases of acute ileus, the mortality rate of the series for all cases must be lowered in addition to the lowering of the mortality of the enterostomy group in that same series, while the mortality of the nonenterostomy group remains relatively unchanged, and the entire series as well as its enterostomy group shows approximately the same percentage of early and late cases as those with which they are compared. When the statistics for acute ileus compiled by Gibson in 1900 are compared with those compiled

TABLE 8.—*Comparison of Mortality by Four-Year Periods in the Same Hospital*

Taken from the Presbyterian Hospital statistics. Note that the largest percentage of improvement is found in the nonenterostomy group.

Cases Referred to	Number of Cases		Mortality Percentage	
	1916-1919	1920-1923	1916-1919	1920-1923
Entire series	60	80	66.6	53.7
Nonenterostomy group	38	44	52.6	34.0
Enterostomy group	22	36	90.9	77.7

by us in 1925 (table 3), the mortality rate of the recent enterostomy group is found to be 3.5 per cent lower than that of twenty-seven years ago, and the mortality rate of the entire recent series is 1.4 per cent lower than that of twenty-seven years ago; but the mortality rate of the nonenterostomy group is 5.2 per cent better than in the old series. Here, then, the mortality rate of the entire series has apparently been lowered chiefly by lowering that in the nonenterostomy group. Any reduction in mortality ascribable to enterostomy has probably been apparent rather than real, and is probably due to the selection of earlier cases. This is a deduction, not a demonstration, because we lack evidence as to the time factor. Our own series at the Presbyterian Hospital shows similar indications (table 8). In the entire four year series the mortality rate has been lowered 12.9 per cent, while the mortality rate of the enterostomy group has been lowered 13.2 per cent At the same time, however, the mortality rate of nonenterostomy cases has been lowered 18.6 per cent. It is evident that the improved mortality rate of the nonenterostomy group is chiefly responsible for the improved mortality rate of the entire four year series, which therefore can be due only in small part to the lowered mortality rate of the enterostomy group.

In analyzing the two four year series reported from the Presbyterian Hospital for early and late cases (table 9, dagger footnote), we find that the first series included 15 per cent more late cases than the second series. We ascribe the improvement in the mortality rate of the second series to early treatment rather than to improved operative technic or to any special procedure such as enterostomy. Moreover, the improvement in the mortality rate of the enterostomy group cannot be justifiably ascribed

TABLE 9.—*Rise in Mortality Due to Late Operation. Group Mortality in Hours After Onset; Comparison by Four-Year Periods**

Taken from the Presbyterian Hospital statistics. Time analysis of the two four-year series shown in table 8.

	Hours Between Onset and Operation				
Percentage Mortality	12	24	48	72	72+
90	**1** ..	..	..	..	..
80	..	..	..	..	.. **38**
70	..	..	..	.. **12**	..
60	..	..	.. **15**	..	..
50	..	.. **11**	..	11	41
40	..	5	5		
30	..				
20	..				
10	.. **1**				

Light faced figures represent series of 1916-1919, inclusive; mortality, 66.6 per cent (1916-1919 series includes about 15 per cent more late cases [after forty-eight hours] than does 1920-1923 series). Bold faced figures represent series of 1920-1923, inclusive; mortality, 53.7 per cent. The numbers show the number of cases in which operation was performed in the twelve or twenty-four hour period.

* Compare these curves with that in table 4.

TABLE 10.—*Analysis of Enterostomy Groups by Hours After Onset*

Taken from the Presbyterian Hospital four year series shown in tables 6 and 7.

Years 1916-1919 Inclusive	Years 1920-1923 Inclusive
1 12 hour case	0 12 hours cases
4 12 to 24 hour cases	5 12 to 24 hour cases
3 24 to 48 hour cases	3 24 to 48 hour cases
2 48 to 72 hour cases	9 48 to 72 hour cases
12 72+ cases.	21 72+ cases
22 cases, mortality 90.9%	38 cases, mortality 77.7%
63.5% of cases were over 48 hours	78.9% of cases were over 48 hours

to the enterostomy procedure until we have decided whether the lower mortality is due to better technic or to earlier operation. In analyzing the two enterostomy groups for early and late cases (table 10), we find that the second group has a larger percentage of late cases than the first group. It should therefore (other things being equal) have a higher mortality rate; we find that it has a lower mortality than the first group. We may, then, justly ascribe the improvement to better technic rather than to earlier operation. Here is statistical evidence indicating that enterostomy has saved some patients with acute ileus who might have

died. It is good evidence, because it was obtained after satisfying the criteria on which a just comparison depends.

SUMMARY

Enterostomy as a treatment for acute ileus has been used in only a small percentage of the total number of cases. It has apparently been reserved as a last resort in the cases in which the patient is the sickest and the condition is most advanced. There is evidence suggesting that this procedure has been effective in lowering the mortality rate, but it has apparently had little influence on the general mortality rate of all cases in a series. If it is to affect this rate (supposing it to be capable of so doing) it must be used in a larger percentage of the total number of cases in a series—in the early as well as in the late groups and more often as a preliminary step to further treatment.

The actual value of enterostomy as a treatment for acute ileus must be determined ultimately by statistical proof. Statistics published up to this time do not afford a satisfactory basis for comparison except in a few instances; those are too few for definite conclusions.

Because of the recognized effect that early opposition has on the mortality of the condition, any series of cases of acute ileus (in order fairly to compare its results with those of another series) must be analyzed into its component groups of early and late cases. This is true also of the enterostomy and nonenterostomy groups in the series of cases of acute ileus. It is hoped that those who are interested in acute ileus will record and publish their series of cases in such a manner that comparisons can satisfactorily be made with a view to establishing definitely the value of enterostomy in the treatment of this condition. The desirability of doing this has been indicated in a recent publication on experimental work by Haden and Orr.[18]

18. Haden and Orr: High Jejunostomy in Intestinal Obstruction, J. A. M. A. 87:632 (Aug. 28) 1926.

A REVIEW OF UROLOGIC SURGERY

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(Concluded from page 154)

PROSTATE

Prostatectomy.—Tengwall [42] performs prostatectomy according to Freyer's technic. The retention catheter is used for bladder drainage. In his first 150 cases no packs were used. One patient died from hemorrhage, and seven had severe postoperative hemorrhage which necessitated reopening the bladder. In the last 100 cases, packs were placed in the prostatic bed, and there was no hemorrhage. The packs were removed in forty-eight hours, and the bladder drained in three or four days. A urethral retention catheter was left in place until the operative wound healed, so that there would be no strain on the wound. In the first fifty cases Tengwall used spinal anesthesia with 3 per cent tropacocain. One patient died from shock. In 140 cases local anesthesia was used to open the bladder, after which the prostatic bed of the bladder was infiltrated with procaine hydrochloride, according to Legueu. In 140 cases in which operation was performed prior to 1918, tests of renal function were not performed; five deaths occurred from uremia. In the last 110 cases in which various tests of renal function were made, there were but two deaths from uremia. Preoperative preparation and forced fluids were emphasized. In all cases examination was made with the stone probe, and if vesical neoplasm was considered, cytoscopy was carried out. If the results of tests by water dilution were good, there being no further contraindication, the patient

42. Tengwall, Ernest: Two Hundred Fifty Suprapubic Prostatectomies for Hypertrophy of the Prostate, Acta chir. Scandinav. **59**:455, 1926; Hygiea **87**:795, 1925; abstr., Ztschr. f. urol. Chir. **20**:173, 1926.

was operated on at once. If results of the water test were poor, drainage was instituted until the renal function improved. If there was no improvement in from four to six weeks, a suprapubic drainage was made, and prostatectomy performed later (in twenty-three cases there was one death). Old age was not a contraindication to operation. Twenty-five patients were more than 80 years old.

In 145 cases only one case of cancer was revealed on histologic examination. In more than half the cases, the urine was badly infected. As the best combatant to this infection Tengwall advised bladder lavage with from 0.5 to 2 per cent silver nitrate solution. In fifty-one of seventy-nine cases the urine prior to operation was infected. Postoperatively, the urine was infected in twenty-seven and normal in fifty-two. Twenty-seven (10.8 per cent) of the patients died. One hundred eighty-eight patients were traced, of whom 180 had improved; two were slightly better, and six were worse.

In seven cases death was due to uremia, in two to hemorrhage, in one to shock following spinal anesthesia, in five to sepsis, in three to pyelonephritis, in four to bronchopneumonia, in three to pulmonary embolism, in one to cerebral embolus and in one to heart failure. The residual urine was tested in 112 cases; in seventy cases there was none; in thirty-six there was from 5 to 50 cc.; in three, 75 cc.; in two, 100 cc., and in one, 150 cc. Postoperative stricture developed in seven cases. In fifty-four (23 per cent) epididymitis developed, in nine of which suppuration occurred; in four cases hernias developed in the wound. Sexual function was noted in seventy-four cases. Thirty patients were impotent before operation; twenty-eight were normal after operation; two lost function after operation; four patients had normal function, but no ejaculation. One 65 year old man impregnated his wife after operation.

Cohen, Dodds, and Webb [43] assert that the nonprotein nitrogen and uric acid content of the blood are the most accurate guides to prognosis. The upper limit of safety for complete operation of prostatectomy was found to be 50 mg. for each hundred cubic centimeters of nonprotein nitrogen, and 3.5 mg. for each hundred cubic centimeters for uric acid. Experience has shown that if figures above these are encountered, it is advisable to perform preliminary suprapubic cystotomy. Enucleation can then be carried out when the figures fall within the limits of safety. The urea content in the blood is much more unreliable than the nonprotein nitrogen and uric acid contents. A high urea content in the blood must always be regarded as a serious sign, but reference to the tables will show that a low urea content cannot always be

43. Cohen, I.; Dodds, E. C., and Webb, C. H. S.: Observations Bearing upon the Operation of Prostatectomy, Brit. J. Surg. **13**:656, 1926.

regarded as an indication of normal renal function. Laboratory data alone should never be relied on, but should always be considered in combination with the clinical state of the patient.

Punch Operation.—Van Houtum [44] summarized the treatment of median bar and vesical neck contracture, mentioning especially Caulk's cautery punch. He treated thirteen patients with Caulk's punch; eleven obtained good results; one remained unchanged, and one died, supposedly from ether anesthesia. Parasacral anesthesia was used in the other cases.

Van Houtum believes that from one fourth to one third of all cases of prostatitis are suited to the punch operation. There are of course many borderline cases in which there is a question of using a punch or performing a prostatectomy. The type of procedure is influenced by the social status of the patient, the experience of the surgeon and the general symptoms. It should be remembered that a prostate may often recede after the urinary stream has been reestablished, and if the punch is not successful prostatectomy can always be performed.

Complications.—Randall [45] points out the failure to realize the extreme importance of some of the so-called lesser postoperative complications. He bases his conclusions on the investigation of a series of 100 cases of prostatectomy in relation to the occurrence of epididymitis. White is quoted as reporting the occurrence of this complication in 82 per cent of fifty cases which he investigated. Eisendrath found the complication in one-fifth of his cases. In Randall's experience it occurred in twenty-three cases in the 500. In four cases it was associated with perineal prostatectomy, and in nineteen the suprapubic operation had been performed. The time of occurrence of the epididymitis varied from five cases preoperatively to six cases after the patient had returned home. As a rule, it occurred just as urethral voiding was reestablished.

Epididymitis was about as common in private patients as in ward patients. There were three deaths in the series; in each case, however, there was some other complication. Healing was generally delayed in cases in which epididymitis developed.

Randall advises support and hot magnesium sulphate compresses, followed later by the application of 5 per cent guaiacol ointment. He believes that careful daily irrigations would do a good deal toward preventing the occurrence of this complication, and he also advises further study of the effects of ligating the vas.

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44. Van Houtum, G.: Erfahrungen mit der Caulkschen Punch-Operation bei kleinen Prostaten, Ztschr. f. urol. Chir. **19**:253, 1926.

45. Randall, Alexander The Rôle of Epididymitis as a Complication of Prostatectomy, J. Urol. **16**:141, 1926.

Prostatic Sarcoma.—Smith and Torgerson [46] reviewed the literature on sarcoma of the prostate and reported eighty-four cases, including one case of their own. Sarcoma of the prostate occurs in adult life in about 65 per cent of cases; in 30 per cent the disease occurs in the first ten years of life. The first symptoms are usually related either to the rectum or the bladder, and are in the form of partial obstruction of the bowel, difficulty in urination or complete retention. Pain, which sooner or later almost always appears, is of two types, that produced by obstruction of the rectum or bladder, and essential pain, severe, lancinating and interfering with rest and sleep, deep in the pelvis and not dependent on urination or defecation. The pain may radiate in almost any direction, but its common course is through the sacrum or down the limbs to the knees. Hematuria is sometimes present, and is due either to ulceration of the bladder or diffusion of the growth throughout the wall. The tumor varies markedly in size. As a rule, on rectal examination, the prostate is found greatly enlarged, particularly in the child. It is usually smooth and firm, although it is sometimes cystic or soft.

The round cell type is the most common; the spindle type is next. Metastasis was noted in about 40 per cent of cases. Distant metastasis is quite rare in early life. Local extension occurred in fifty-nine cases (72 per cent). The bladder was most often involved, extension to this structure occurring in forty-one (70 per cent) of the cases. In twenty-three cases (40 per cent) the urethra was involved, and in eleven cases the rectum. Treatment is not satisfactory, and the outlook is poor. All the patients in this series died from the disease.

BLADDER

Tumors.—Simon [47] reports seventy-three operations for carcinoma of the bladder performed on fifty patients. Twenty-four of the patients (48 per cent) are alive, and twenty-six (52 per cent) are dead. In twelve of the latter a radical operation could not be performed. Twenty-two of the twenty-four living patients had papillomatous tumors; only two tumors were of the infiltrating type. Eight of the twenty-six patients who died had infiltrating tumors.

According to Judd,[48] the results of radical operation for carcinoma of the bladder have not come up to expectations; they are about the same as those obtained in radical operations for carcinoma of the breast, stomach,

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46. Smith, R. R., and Torgerson, W. R.: Sarcoma of the Prostate; Report of a Case with a Digest of the Literature, Surg. Gynec. and Obst. **43**:328, 1926.

47. Simon, L.: Die Behandlung und Prognose der malignen Blasengeschwülste, Beitr. z. klin. Chir. **136**:565, 1926.

48. Judd, E. S.: The Treatment of Carcinoma of the Bladder by Radical Surgical Methods, J. A. M. A. **87**:1620 (Nov. 13) 1926.

colon and other organs. Seven hundred and eight cases of carcinoma of the bladder from the Mayo Clinic are reviewed. Two hundred and thirty-eight of the patients are known to be living. Ninety-four have lived three years or more; sixty-two, five years or more; and five, ten years. Fifty of 298 patients who had surgical treatment alone or surgical treatment with radium lived more than five years, and twenty-eight of 308 patients who had other forms of treatment lived more than five years.

In 261 of the 272 patients traced, the tumors were graded according to the degree of malignancy. Of twenty-one patients with tumors graded 1, sixteen are living; three have died since leaving the hospital; of eighty-one with tumors graded 2, thirty-eight are living; thirty have died since leaving the hospital; of 103 with tumors graded 3, twenty-seven are living; sixty-two have died since leaving the hospital; of fifty-six with tumors graded 4, sixteen are living; thirty-two are known to have died after they reached home. Five patients lived ten years; in one malignancy was graded 3; in two it was graded 4; and in two, 1.

Of the 708 cases, 102 were too far advanced, or the patient was in too poor general condition to warrant any kind of treatment. The average duration of the disease in the 102 cases was thirty-two months, and the average length of life after the examination was eight and nine-tenths months at the time of publication of the report. Forty-three of the 708 patients were treated by radium and fulguration, and this group showed the highest average length of life after treatment, which was thirty-six and seven-tenths months. Fourteen were treated with radium and cautery, and the average duration of life afterward was ten and seven-tenths months, which was but little longer than in those who were not treated. But this figure is deceiving, for some of the best results have been obtained by the use of the cautery in cases in which the disease seemed well advanced. Seventy-nine patients were treated with radium combined with excision or resection, and they lived, on an average, twenty-one and fifty-six hundredths months afterward. Of the 708 patients, 219 received surgical treatment alone, and the average postoperative length of life was eighteen and fifty-seven hundredths months; nineteen of the 708 were treated by surgical diathermy. The time since treatment is too short to determine what the ultimate results will be. However, the immediate results in some of the cases in which the disease was extensive have been extremely gratifying.

From the standpoint of treatment, Judd divides tumors of the bladder into three groups. The first group should include cases of benign tumor. Most of such tumors are papillomas and should be treated by endoscopic methods. The second group should include all malignant cases in which the process is still confined to the bladder and all cases in which the diagnosis is questionable. The treatment indicated is

radical removal of the growth and reconstruction of the bladder, if that is possible. In the third group should be placed cases in which the malignant growth is too extensive for removal. If the lesion is confined to the bladder it can be removed by radical operation, but if it has extended to the perivesical tissues, fixing the bladder firmly to the prostate and seminal vesicles or to the other organs in the pelvis, it is not advisable to attempt to remove it. For growths too extensive for radical removal, surgical diathermy is the best procedure. Such treatment should be carried out in cases in which the carcinoma of the bladder is less extensive, if there is some contraindication to the radical operation, such as cardiac or renal disease.

Ureteral Transplantation.—Chute [49] believes that more radical measures must be adopted, such as transplantation of the ureters, if better results are to be secured in cases of carcinoma of the bladder. In eighty-six of 170 of Chute's operative cases, the patients are known to have died as a result of operation or disease; eighteen others had recurrences. Another fact is taken into account in considering the results in Chute's cases; that a large percentage of the patients, nearly one-third, on whom an open operation was performed and who are not known to have died, had been treated for so-called simple papillomas, often quite small, a number of years previously. Such cases are at present not subjected to open operation, but are more satisfactorily treated by fulguration.

Chute reports five cases of cystectomy for carcinoma; all the patients died. In one case, a large pocket of pus was found deep in the pelvis near the right ureter. In the second case, death was due to pyelonephritis and sepsis from a sloughing pelvic cavity. There was necrosis of the sigmoid at the site of ureteral transplant in the third case. Death resulted from embolic pneumonia in the fourth case. Necropsy was not obtained in the last case.

In spite of the poor results obtained in his cases, Chute firmly believes that total cystectomy, either proceded or followed by transplantation of the ureters, is the best way of dealing with extensive infiltrating tumors of the bladder, stubborn recurring growths of the papillomatous type and growths which involve the outlet of the bladder. He says that prostatocystectomy is probably the operation of choice for men. He discusses the Coffey technic of implantation of the ureters into the rectum.

Hinman [50] believes in transplanting the ureters into the bowel in a small group of cases. Although the method is now usually employed

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49. Chute, A. L.: Ureteral Transplantation in Bladder Carcinoma, J. A. M. A. **87**:1613 (Nov. 13) 1926.

50. Hinman, Frank: The Indication of Nephrostomy Preliminary to Ureterorectoneostomy, J. A. M. A. **86**:921 (March 27) 1926.

only as a last resort, if freed from some of its immediate and remote dangers it could be applied to a larger number of cases. The procedure is most common in cases of exstrophy of the bladder, but even in this type of case it is not entirely satisfactory, as after the transplantation further urologic study of the part is not possible; in one case after transplantation a stone blocked the ureter and could not be reached through the rectum. Hinman believes that a surgical victory over this defect must include a restoration of natural relationships, that is, repairing the cleft symphysis as one would repair a cleft palate.

Ureteral transplantation is indicated in certain acquired conditions, such as extensive vesical carcinoma, necessitating total cystectomy. Transplantation should also be performed in cases of extensive tuberculosis of the bladder, if an infected kidney has been removed, and the patient is suffering from frequency and pain. Unless this is done the bladder contracts the ureteral orifice, causing dilatation of the remaining kidney. In several cases, hydronephrosis was found in the remaining kidney without any sign of tuberculosis.

Carcinoma of the uterus may necessitate radical resection, and sometimes the ureters are involved. Usually the surgeon ties the ureter and then removes the kidney. If uretero-intestinal anastomosis were a safe procedure more radical measures might be undertaken. The rectum is a better site for the transplant than the skin, since it gives continence. The difficulties of a successful transplant of the ureter are inherent and technical, and immediate and remote. The inherent objections are putting the ureters in an infected field in which urologic studies cannot be carried out. The technical ones, such as the spread of infection, peritonitis and fecal fistula, may be overcome. The immediate dangers result from the edema at the site of the transplant, which causes ureteral obstruction and ascending pyelonephritis or anuria.

To insure satisfactory renal function Hinman advocates a first-stage, preparatory nephrostomy, followed subsequently by ureteral transplantation. Lumbar drainage permits direct treatment and free drainage of the kidney. At the same time the ureter could be treated after the transplantation by irrigation and dilatation. Hinman has used this procedure with success in two cases.

Injuries to the Bladder.—Peacock and Hain [51] discuss the problems associated with injuries of the urethra and bladder. Straddle falls, although a common cause of such injuries, are not so common as in the past. The advent of certain industrial hazards and the automobile have given crushing injuries a larger place in the etiology of these conditions. Industrial accidents account for 55 per cent of the cases reported

51. Peacock, A. H., and Hain, R. F.: Injuries of the Urethra and Bladder: A Study of Thirteen Cases, J. Urol. **15**:563, 1926.

by these authors. Automobile accidents were etiologic factors in 18.7 per cent of the industrial and 50 per cent of the nonindustrial accidents. Crushing injuries accounted for 46.4 per cent of the cases and straddle injuries only 17.4 per cent. Fracture of the pelvis was common, associated with injury of the urethra, although the urethra was uninjured in a majority of fractures of the pelvis. Gunshot wounds are rare in civil life. Automutilation is seldom seen except among insane patients.

It is not surprising that 89 per cent of the injuries of the bladder and urethra occurred in men, possibly because of their greater activity, their occupational hazards and the close anatomic relation of the urethra to the pubic arch. The urethra is firmly fixed to this bridge of bone, and any fracture or displacement of the latter is likely to tear across the urethra. In women the urethra is shorter, less firmly attached, and moves with the anterior vaginal wall.

Latent symptoms in order of occurrence are: dysuria, frequency of urination, loss in strength, urinary infection, impairment of sexual power, hematuria, periodical retention and backache.

Immediate operation, such as suprapubic cystotomy, retention catheter, suprapubic cystotomy and laparotomy, and suprapubic puncture, may be required; while late operations are dilatation with sounds or bougies, combined suprapubic cystotomy and external urethrotomy.

The average time in the hospital is sixteen weeks. The average period of disability is twenty-three months.

The automobile is directly responsible for one-third of these injuries. The industrial disability phase is important as it involves extensive hospitalization, loss of time, and only 33 per cent of the patients can be called cured. Fifty-five per cent of the patients were entitled to industrial compensation.

Exstrophy.—Mayo and Hendricks [52] report sixty-six cases of exstrophy of the bladder in which operation was performed at the Mayo Clinic between 1901 and 1926. Forty-eight of the patients are living, and eighteen are dead. Plastic operation alone for closure of the bladder was performed in eight cases; transplantation of both ureters in forty-six cases; transplantation of one ureter in nine cases; exploration with closure because of hydro-ureter or hydronephrosis in two cases; transplantation of one ureter to the hepatic flexure in one case; suprapubic cystostomy for excision of carcinoma of the bladder in one case; the Moynihan operation in two cases, and the recent Coffey operation in three cases. In two of these both ureters were transplanted simultaneously; and in the other, one ureter was transplanted. Eleven patients, 16.6 per cent, died in the hospital; they lived from two to

52. Mayo, C. H., and Hendricks, W. A.: Exstrophy of the Bladder, Surg. Gynec. Obst. **43**:129, 1926.

nineteen days after operation. Five patients died from peritonitis; and six died from urinary infection, hydro-uretero-nephrosis and uremia. Complete statistics were obtained concerning twenty-nine patients. Twenty-nine were satisfied with the results. Two had poor control of urine by rectum. The period between emptying the rectum varied from two to five hours by day and from three to eight by night. One patient was incontinent. Bowel movements varied from five to six times a day to once or twice. The general health of the patients was invariably good; most of them had gained in weight, had good appetites and slept well. Six of the twenty-nine patients are living from one to three years after operation; ten are living from three to six years; ten are living from six to twelve years, and three are living from twelve to fifteen years.

Vesico-ovarian Fistula.—Fronstein and Sserdjukoff [53] describe a case of vesico-ovarian fistula in a woman, aged 38. The rupture of purulent abscesses in the parametrium, of suppurating dermoid cysts, of ectopic pregnancy and of suppurating tubal abscesses, into the bladder is not infrequently described, but according to these authors there is no literature on vesico-ovarian fistula, and in this respect their case is unique.

The clinical picture of the rupture of any pelvic abscess into the bladder begins with frequency and dysuria, due to inflammatory infiltration of the wall of the bladder. At this stage cystoscopy reveals bullous edema of the wall at the affected site. With the concurrence of rupture, the urine becomes loaded with pus. There is improvement in the subjective symptoms, the temperature falls, and the cystoscope shows a point of rupture usually of small diameter, surrounded by edematous mucosa. Frequently pus may be found trickling from the point of rupture. The course of the disease then becomes intermittent. If there is good drainage of the pus into the bladder, the subjective symptoms and the temperature are more favorable, and there is little pain. With occlusion of the point of rupture, from time to time, the urine becomes clear, but there is pain and fever with dysuria. Frequently and early in the course of the disease, an ascending infection of the kidneys develops.

Conservative treatment includes lavage of the bladder, drainage of the pelvic abscess by vaginal or abdominal routes or the attempt to clear up the inflammatory mass by nonspecific protein therapy. With the subsidence of the pelvic infection under such treatment the vesical fistula frequently heals, but this end-result is by no means certain. Radical excision of the perforating mass together with the vesical fistula, and the suture of the bladder, although attended by greater operative risk, are necessary when conservative methods fail.

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53. Fronstein, R., and Sserdjukoff, M.: Zur Frage der Ovarial-Blasenfisteln, Ztschr. f. urol. Chir. **19**:102. 1926.

In the authors' case a large ovarian abscess on the right side had ruptured into the bladder by way of the right broad ligament. Bilateral salpingitis, suppurative oophoritis and myometritis were present. After one and a half months of conservative treatment, including repeated intravenous injections of methenamine solution, the fistula persisted and symptoms were only slightly improved. Laparotomy was performed with resection of the adnexa, of the abscess and of the fistulous tract, which was found to be connected with the ovary. The bladder was sutured, the abdomen closed and an indwelling urethral catheter inserted for six days. The convalescence was marked by a probable infarct of the stomach, clinically suggested by nausea, coffee-ground vomitus and pain in the region of the greater curvature. This cleared up shortly, and the patient recovered satisfactorily. On examination after five months there was no marked inflammatory process in the bladder or vagina.

[ED. NOTE.—The vesico-ovarian fistula described here is unusual, although it is remarkable that more cases have not been noted. The danger of radical operation by laparotomy is obviously that of spreading the infection into the general peritoneum. Fronstein and Sserdjukoff used ether to wash out the pelvis at the completion of the operation before closure. When dealing with large pelvic masses which cannot be properly drained through the vagina, and when conservative methods do not result in prompt improvement, probably the best recourse will be that of radical resection as described.]

Infections of the Bladder.—Dickson [54] reports the case of a woman, aged 37, who had suffered from a urinary infection for many years. In 1916, sporelike bodies together with other organisms were found both in the urine from the bladder and in that from the ureter. Corresponding with this fungus-like organism, a pleomorphic organism was sometimes found in the cultures, but it was grown with extreme difficulty and soon died out in the cultures. Similar observations were made from time to time, and on May 6, 1925, a small shred or clump of large irregular cells, containing numerous vacuoles, was found. When these were examined wet, and faintly stained with methylene blue and other stains, they contained apparently structureless, clear, rounded or slightly oval bodies or inclusions, which strongly suggested the so-called Michaelis-Gutmann bodies of malakoplakia.

Fresh, wet preparations of the centrifugalized deposit of the urine showed considerable numbers of large cells containing the cell inclusions already mentioned. These inclusions varied greatly in size, from 3 to 10 microns or even more. When still fresh, they appeared absolutely

54. Dickson, W. E. C.: Demonstration of a New (?) Mycotic Parasite Found in the Bladder in a Case Resembling Malakoplakia, Proc. Roy. Soc. Med. **18**:54, 1925.

structureless, homogeneous, rounded or slightly oval, and occasionally sausage-shaped, refractile and slightly greenish, and with a definite-clear-cut outline. In a few hours little mycelial filaments began to sprout, and soon every cell appeared like germinating peas, the thickness of the filaments varying from 1 to 5 microns, or even more. The growth was so rapid that a slow, snakelike writhing movement was seen distinctly on two occasions. Yeastlike budding was also present. As the original cells of the exudate gradually disintegrated, numerous spherical bodies were set free and passed into the fluid; these varied in size up to 12 microns, or even more, in diameter.

Patches resembling malakoplakia were seen in the bladder through the cystoscope, and specimens were snipped out for examination. Wet preparations of these exhibited the same curious organism in many of its forms. The most numerous were the perfectly spherical, hyaline, structureless bodies which can best be described as resembling little drops of oil.

Blanc [55] states that in the treatment of vesical tuberculosis with methylene blue the dye must be chemically pure, entirely dissolved and filtered before using. Satisfactory solutions make a homogeneous blue stain on linen, while inferior solutions give a pale blue tint which may be surrounded with unstained areas. A 1:100 solution is made in normal sodium chloride solution and must be tepid on instillation. One may begin with from 5 to 10 cc., but if bladders are intolerant the amount may be reduced or a weaker solution may be used at the start, after which the concentration and amount may be increased gradually. In the presence of strong pyuria, the bladder should be irrigated with a 1:4,000 to 1:8,000 oxycyanide of mercury solution before instillation. A Nélaton catheter is used for women, and a small Guyon catheter for men. After such an instillation the patient should move about, since by so doing he may be better able to retain the instillation.

Herpes of the Bladder.—According to Schiffmann,[56] there has been much confusion in the literature as to just what has been designated herpes of the bladder. Lipschütz has shown by experiments in vaccination that herpes genitalis is an infectious process of the skin and mucous membrane. It is closely related to herpes febrilis and herpes zoster, but must be differentiated from them.

Schiffmann reports the case of a woman, aged 31, who had a general eruption about the genitals, which proved to be a typical herpes. The vagina and mucous membranes were normal. Later the genitals were completely covered and edematous. Complete retention developed, and

55. Blanc, Henry: La technique des instillations vésicales de bleu de méthylène dans la cystite tuberculeuse, J. d'urol. **20**:144, 1925.

56. Schiffmann, Josef: Herpes der Blase, Ztschr. f. urol. Chir. **19**:342, 1926.

catheterization was necessary. The urine contained red blood cells and small clumps of pus. Culture of the urine was sterile. This condition persisted for four weeks and a half, during which time the bladder was washed. There was no cystoscopic examination; nevertheless, Schiffmann believes that the case is herpes of the bladder because of the outer manifestations and a definite resistance encountered at the vesical neck, which was attributed to blebs. The retention was reflex, probably because of the pain from the herpes genitalis. As the latter healed, normal urination was reëstablished.

Du Bois [57] submits a case of "herpes zoster of the bladder," the diagnosis being based on the cystoscopic data of typical herpetic vesicles, coincident with a typical herpes zoster of the buttock, of the same side. It is noteworthy that the nerve supply of the buttock and of the base of the bladder is the same; that is, from the sacral plexus. He believes that the diagnosis is further confirmed by the more or less rapid disappearance of both the cutaneous and mucosal lesions at the same time, without any particular treatment, other than by mild urinary antiseptics and a local application of methylene blue to the external lesions.

Nishiura [58] states that varices of the bladder are not common. Prior to the advent of the cystoscope, diagnosis could be made only at necropsy. The condition has been designated "bladder hemorrhoids." For the most part it is only an expression of venous stasis in the pelvic organs. Certain observers have noted that bleeding seldom occurs, but sometimes with disturbed menstruation the varices may cause vicarious bleeding from the bladder. A case is cited in which there was hematuria, and cystoscopy revealed varicose veins of the bladder and a coagulum at the base. Since the patient had hemorrhoids, it was assumed that the two were associated. In treating these patients the retention catheter, ergotin, chlorcalcium intravenously or by mouth, or the high frequency current may be used. Should these be inadequate, suprapubic cystotomy is indicated.

URETHRA

Tuberculosis.—Richter [59] reports the following cases: A man, aged 47, had had symptoms of renal tuberculosis for one and a half years, and recently tuberculous epididymitis had developed. There were no subjective symptoms of urethral stricture, but following the passage of small sounds acute miliary tuberculosis appeared, and the patient died.

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57. Du Bois, F. E.: A Case of Herpes Zoster of the Bladder, J. Urol. **15**:583, 1926.

58. Nishiura, Koitchi: Les varices, vesicales, Acta Dermat. **6**:325, 1925; abstr., Ztschr. f. urol. Chir. **19**:359, 1926.

59. Richter, Sölver: Zur Kenntnis der Harnröhrentuberkulose, insbesondere ihrer strikturierenden Form, Acta chir. Scandinav. **59**:237, 1925; abstr., Ztschr. f. urol. Chir. **19**:82, 1926.

The second case was that of a man, aged 32, with definite signs of advanced urogenital tuberculosis. He also had a small stricture of the urethra which admitted only a filiform. In the process of dilatation acute miliary tuberculosis developed, from which the patient died.

The third case was that of peri-urethral phlegmon with sepsis. At operation it was found that tuberculosis of the urethra had been the primary lesion. Tuberculosis of the seminal vesicles was also found; this appeared to have developed more recently.

From these cases Richter concludes that tuberculosis of the urethra may become extensive without causing marked symptoms. For this reason, in any case of urogenital tuberculosis, one must bear in mind the possibility of tuberculous urethral stricture. Richter warns against the dilatation of tuberculous urethral strictures with sounds. He recommends operative exploration of the kidneys or ureters instead of cystoscopic investigation. He mentions further thirteen cases of urogenital tuberculosis with accompanying urethral strictures. In nine of these the primary focus was removed by nephrectomy, and in eight of the nine the urethral tuberculosis cleared up. Thus he concludes that urethral strictures of tuberculous origin should not be dilated with sounds, but should be treated by removing the primary focus, usually the kidney.

[ED. NOTE.—Tuberculous stricture of the urethra is an unusual lesion and is scarcely mentioned in textbooks on urology. When it does occur it is always secondary to genito-urinary tuberculosis elsewhere. For this reason one would assume that in many cases if the focus above the urethra, usually the kidney, is cleared up by operation, the urethra would tend to heal just as the bladder does. Thus Richter's conclusions seem logical and in accordance with accepted pathologic principles.]

Anomalies.—Ritter [60] notes that there are but fifty-seven reported cases of double urethra. He reports a case in a boy, aged 2½, suffering from partial incontinence. Examination revealed a double urethra, one normal through the penis, and one an epispadic structure. The latter had no sphincter muscle, a condition which accounted for the incontinence. Treatment consisted of dilating the normal urethra and successful resection of the epispadic urethra. Histologic examination of the latter failed to reveal any evidence of sphincter muscle, but many nerve fibers were found. Ritter believes that the supernumerary urethra came from the same anlage as the normal urethra and that it grew out with the penis, but remained in communication with the bladder.

Baradulin [61] noted thirty-nine cases of double urethra in the literature, three of which were reported by Russian observers (Koltchin,

60. Ritter, O.: Beitrag zur Kasuistik und Behandlung der durchgehenden, epispadischen Urethraverdoppelung, Ztschr. f. urol. Chir. **20**:5, 1926.

61. Baradulin, G.: Ein Fall von doppelter Urethra, Urologya **2**:3, 1925; abstr., Ztschr. f. urol. Chir. **20**:120, 1926.

Dedjurin, Fronstein). In Baradulin's case the extra urethra lay above the normal urethra, the separation being from 6 to 7 cm. from the external meatus. He cauterized the septum between the two channels to make a single passage.

Polyps.—Lloyd[62] reports thirteen cases of urethral polyps. They are frequently associated with chronic posterior urethritis and chronic prostatitis. This was noted in most of the cases reviewed. The chronic infectious process may be of long standing, preceding the formation of the polyps by many years. The polyps disappeared in one of Lloyd's cases after the patient had been suitably treated for prostatitis. The polyps in most cases were situated at or near the orifices of the prostatic or ejaculatory ducts, and in many instances showed evidence of an inflammatory origin. They resemble closely polyps found in the nose, which are generally conceded to be due to inflammation. Lloyd believes that inflammation is responsible for most urethral polyps. Treatment is generally ineffective, if the underlying disease in the prostate and vesicles is not treated at the same time.

TESTICLE

Tumors.—According to Handfield-Jones,[63] the gross appearance of chorionic carcinoma of the testicle depends largely on the amount of ordinary teratomatous tissue present. If a recognizable quantity is present, the picture will show typical signs, such as small cysts and areas of blue translucent cartilage. The area of chorionic carcinoma has the appearance of a blood clot. Plum-colored areas are seen, practically homogeneous throughout, except for small, white, friable areas of necrosis. The elements found in this type of carcinoma are briefly: (1) syncytium, (2) Langhans' cells, (3) chorionic wandering cells, and (4) masses of blood and fibrin. These elements are destribed in detail.

Two types of tumor are noted. These are best described by Frank as follows:

> In the typical group the characteristics of the chorionic epithelium, which appears in the first stage of gestation, are represented with few or no variations. They show well-developed continuous syncytial masses of irregular multinucleated strands and branching protoplasmic buds, and with a more or less well-developed ground work of numerous transparent polyhedral cells of the nature of the Zellschicht (viz., Langhans' cells).
>
> In the atypical group, nearly everywhere the epithelium has lost its peculiar normal grouping, and occurs only in isolated cells which may be of various forms. The cell masses which form the surface and inner layers of the decidua basalis in

62. Lloyd, V. E.: A Clinical Study of Polypi of the Prostatic Urethra and Their Relation to Chronic Prostatitis, with a Report of Thirteen Cases, Guy's Hosp. Rep. **75**:234, 1925.

63. Handfield-Jones, R. M.: Chorionic Carcinoma in the Testicle: With a Report of a New Case, Brit. J. Surg. **13**:606, 1926.

a hydatidiform mole may be taken as an example or pattern of these forms. These cells rarely show the habitus of the delicate transparent, membranous, and often sharply circumscribed Zellschicht element with regular oval nuclei, frequent mitoses, and glycogenic cell protoplasm. More often they are compact, more deeply staining, and very irregularly shaped cells, with nuclei varying greatly in size, sometimes attaining properties, which allow the recognition of their syncytial character. These elements may form multinuclear aggregates, but in many cases no large syncytial masses occur.

Metastasis is common by the blood stream, but the lymphatics are also usually involved early and extensively.

In the present stage of knowledge of the origin of the teratomas, it is not possible to be assured of the finer details of their derivatives.

Treatment for Cancer.—Roentgen-ray and radium treatment for diseases of the bladder and prostate is discussed in the Section on Urology of the Royal Society of Medicine. Finzi [64] outlines the methods of applying radium in the treatment of patients with diseases of the prostate and bladder. Large dosages are applied to the skin; about 500 millicuries. The urethral route is dangerous, as effective dosage would cause sloughing. The method of embedding radium in the prostate has been used extensively in Brussels. The radium in platinum needles is introduced through an incision in the perineum. The improvement in technic in recent years has consisted in the use of a number of needles instead of one or two large tubes, with the result that the irradiation is much more homogeneously distributed. The results in such cases are often satisfactory, and careful observation will be necessary to determine whether the results are as good as surgical procedures. In inoperable cases, the roentgen ray or radium, or both, should always be tried. In cases of malignant disease of the bladder, operation should be performed, if possible. If not, it is worth while to irradiate, even as a palliative measure. Papilloma of the bladder should be treated by other methods, unless too much surface wall is involved.

Browne-Carthew [64] discussed the treatment of diseases of the prostate and bladder by applying radium through the rectum. He used 50 mg. of the bromide of radium with an activity of 24,000 to 25,000 as measured and estimated in the Institut de Radium, Paris. The radium was in a tube which had been specially made for the treatment of a recurrent malignant lesion of the rectum. It had occurred to him that with this tube radium could easily be applied to the prostate and base of the bladder. He had had at one time under treatment and observation twelve or more patients with simple enlargement of the prostate. The patients had found that nocturnal frequency was diminished and that they passed a

64. Finzi, N. S.; Browne-Carthew, R. H.; Morson, A. C., and Kidd, Frank: Discussion on Radiotherapy and X-ray Therapy in Diseases of the Bladder and Prostate, Proc. Roy. Soc. Med. **18**:15, 1925.

better stream. In some of these cases, on palpation by way of the rectum, the prostate seemed to have been softened by the treatment, but such palpation was indefinite.

Morson [64] states that in malignancy of the prostate, as in every other organ of the body, the only chance of eradication of the disease is by means of surgical intervention. He has been experimenting recently with a new electrical instrument on the lines of the cautery-punch of Young, but his treatment of tumors of the prostate by this means has been disappointing. In about 1 per cent of cases, diathermy was of value. If the obstruction in the prostatic urethra was of fibrous tissue, the use of this electrical treatment offered a more hopeful prognosis.

Kidd [64] states that tumor in the bladder and prostate is a local disease in its earliest manifestations. If seen by the surgeon early, the best chance of cure is still to be obtained with the knife. Kidd feels that the improvements in the results of the modern operation on the prostate and bladder are not appreciated. He reports 129 cases of prostatectomy with only six deaths; twenty-one of thirty-five patients on whom subtotal cystectomy was performed for carcinoma of the bladder during the last fourteen years are apparently cured. He feels that the public is in danger of forgetting that operation can cure cancer in a fair proportion of cases. Physical agents, such as radium, the roentgen ray and diathermy, should be considered as adjuvants to surgical methods, not substitutes.

Summarizing his cases, Kidd states that eight were cases of carcinoma of the prostate. Four were cases in which no operation of a radical type could be carried out. All four of these patients are dead. In none did the roentgen-ray treatment show a favorable effect, and in two it appeared to make the patients ill and to hasten death. In three cases he removed what seemed to be simple senile growth of the prostate. Microscopic examination of the prostate showed early malignant changes. These three patients were exposed to the deep roentgen-ray treatment and are now alive and well. Nevertheless, the results have not been better than in a considerable number of similar cases in which patients showing these early malignant changes were operated on, but were not given the deep roentgen-ray treatment. Recurrence is not the rule in these cases, whether or not this treatment is carried out.

Kidd concludes that by deep roentgen-ray treatment little, if anything, is accomplished in malignant disease of the prostate, except as a prophylactic following operation. He believes that the cells of a malignant tumor of the bladder are not destroyed. After two treatments the rays seem to lose their lethal effect, presumably because the surviving cells become more resistant to the roentgen ray. Roentgen-ray treatment will undoubtedly prolong life for one or two years and may render the patient's life more comfortable, if burns are avoided, as for a time it may relieve or even abolish pain, hemorrhage and frequency of micturition.

It may be expected that deep roentgen-ray treatment will destroy a certain number of cancer cells. The vascularity of a tumor will be lessened, and the fibrous tissue around the tumor increased. The treatment also may produce a certain degree of active immunity. On the other hand, its immediate effect when applied over the abdomen is to cause protein shock, destruction of red blood corpuscles and depression of renal, suprarenal and hepatic function, which debilitated patients cannot incur without risk of life. If the treatment is persisted in too long it may render the cancer cells more resistant and even hasten metastasis; if it is too frequent or too intense it may produce severe, painful gangrene of the abdominal wall and sacral region. This occurred in 8 per cent of Kidd's cases.

According to Thomson-Walker,[65] tumors of the bladder are successfully removed by operation. In 119 cases of simple papilloma he employed transurethral electrocoagulation. None of the patients died, and 74.4 per cent were free from recurrence for from one to eleven years. Operation (excision) was performed in 142 cases, with a mortality of 3.49 per cent, and 65.74 per cent were free from recurrence. In 126 cases of malignant growth in the wall of the bladder, resection was performed; the mortality was 6.34 per cent, and 66.3 per cent were free from recurrence. On the whole, the author was satisfied with the results, but in certain cases diathermy or operation failed, owing to the persistence or recurrence of the growth or because the measures were unsuitable in the advanced state of the growth, because of the septic or contracted state of the bladder, and disease of the kidney, or because of the feeble general state of the patient. Of the 491 cases of tumor of the bladder, 118 were unsuitable for radical operation, in most instances because of the extent of the growth. In view of the successful results obtained by irradiation elsewhere in the body, there seemed to be some prospect of success here also, but the author's experience in a few cases did not encourage him to hope that the surgeon would obtain assistance in operating from preparatory treatment by irradiation; rather, his opinion was the reverse. Neither were his experience and postoperative treatment of growths of the bladder, either by the gamma rays of radium or by hard roentgen rays, encouraging. He noted that mild urinary infection was sometimes converted into severe cystitis, and in other cases there was delayed healing of the wound or tendency of the wound to break down after such postoperative irradiation. His opinion was, therefore, that irradiation in the immediate postoperative stage was not suitable for growths in the bladder. If the growths were inoperable or if growths had recurred after operation, the result with hard roentgen rays was sufficiently encouraging to warrant a more extended trial. The

65. Thomson-Walker, John: Radiology in Urinary Surgery, Brit. M. J. **1**:656, 1926.

only objection to irradiation in such cases is the local reaction, but this does not counteract the good accomplished.

Thomson-Walker believes that radical operation for cancer of the prostate is too severe, and that irradiation is more satisfactory. Forty-eight of his patients who had carcinoma of the prostate were treated by hard roentgen rays. The treatment was carried out by twelve roentgenologists, seven of whom were in London; one patient was treated at Erlangen. General reaction was noted in eighteen of these cases; it was severe in seven. Local reaction occurred invariably, although it differed considerably in cases which otherwise appeared similar. At the time of the report twenty-four of the series of forty-eight patients were dead, four were seriously ill, nine were unchanged or slightly worse, six were improved, four were well, and of one there was no record. Eight of the twenty-four patients who died lived six months after the first exposure; seven patients lived from six to twelve months; six from twelve to eighteen months; two from eighteen months to two years, and one two years and seven months. The condition at the time of irradiation of the patients in this series and in those who remained seriously ill was: advanced growth with the patient failing in twelve; large growth but general condition good in nine; and early growth and general condition good in seven. In forty-two of the forty-eight cases full notes were made of the changes in the prostate. In twenty-two, following irradiation, there was no decrease in size, hardness or fixity of the growth; in fifteen there was improvement, which did not continue; and in five there was improvement which continued. The improvement in some of the cases was slight, but in others pronounced. The hard prostatic mass appeared to melt away, and left only hard nodules, although after a period varying from two to six months the induration reappeared. In five cases, however, the growth disappeared entirely after irradiation. One of these patients, an old man, died six months after treatment from gradual failure, but there was no recurrence of the growth. The other four were alive and without recurrence, the period since treatment being eight, twenty-one, forty-one and forty-five months, respectively. One of these cases was of rapidly growing endothelioma; the others were of the ordinary scirrhous type.

Waters [66] states that the best treatment for superficial papillary carcinoma of the bladder is a combination of deep roentgen ray and radium applied directly to the surface of the growth. Most tumors can be destroyed by this method with a minimal amount of injury to the bladder or bladder mucosa.

Infiltrating carcinoma, if operable, should be treated by radical resection. Twenty-five per cent of the infiltrating growths in Waters' series

66. Waters, C. A.: Deep Roentgen-Ray Therapy in the Treatment of Carcinoma of the Bladder, J. A. M. A. **87**:1618 (Nov. 13) 1926.

were in situations that rendered them inoperable, or they were so extensive that radical removal was impossible. In such cases, when it is possible to apply radium directly to the growth, both radium and deep roentgen-ray therapy should be given a trial, for in a certain number of the cases favorable results can be obtained by this method alone. In cases in which this procedure does not yield the results hoped for, or in cases in which one feels that the growth is sufficiently localized to warrant implantations of radium needles, the bladder should be opened suprapubically, and screened radium needles implanted throughout the growth. If the growth is so extensive that a total of more than 2,500 mg. hours is necessary, in order to destroy the cancerous areas thoroughly by implantations, this method should not be considered.

Tuberculosis.—Ullmann [67] has noted results in forty-one cases of tuberculosis of the testicle, seminal vesicles and prostate treated with the roentgen ray since 1913. In most cases the disease was controlled. The success was questionable so far as the local process was concerned. No injurious results were noted. Some areas of induration were absorbed and fistulas closed. A number of patients died from general tuberculosis. Ullmann states that well localized growths, especially in the testicle, should be removed, and then roentgen-ray treatment should be given.

Diagnosis.—Fryszman [68] believes that the cystoscope gives little information concerning the shape and form of diverticula. Because of the small field of cystoscopic vision, it is hard to estimate the size and shape of the stones. This information may be obtained from summing up numerous small fields, but even then only a relative idea is obtained. In some cases a simple roentgen-ray examination is of no value because the shadow of the stone overlies that of the spine. Fryszman has overcome this by making roentgenograms with his patients uniformly in the Trendelenburg position. In examination of diverticula, he used contrast cystography in the Trendelenburg position. In this position, the pressure of the adjacent organs is removed, and the true size of the diverticulum is demonstrated. In studying growths of the bladder he demonstrates what he terms "pneumocystography," which consists of placing the patient in the Trendelenburg position and inflating the bladder with air. He gives several illustrations which show fair results.

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67. Ullmann, K.: Röntgentherapie bei der männlichen Genitaltuberkulose, Wien. med. Wchnschr. **75**:2879, 1925.

68. Fryszman, Aleksander: Ein Beitrag zur Röntgendiagnostik der Blasensteine, Divertikel und Tumoren der Blase, Ztschr. f. Urol. **20**:321, 1926.